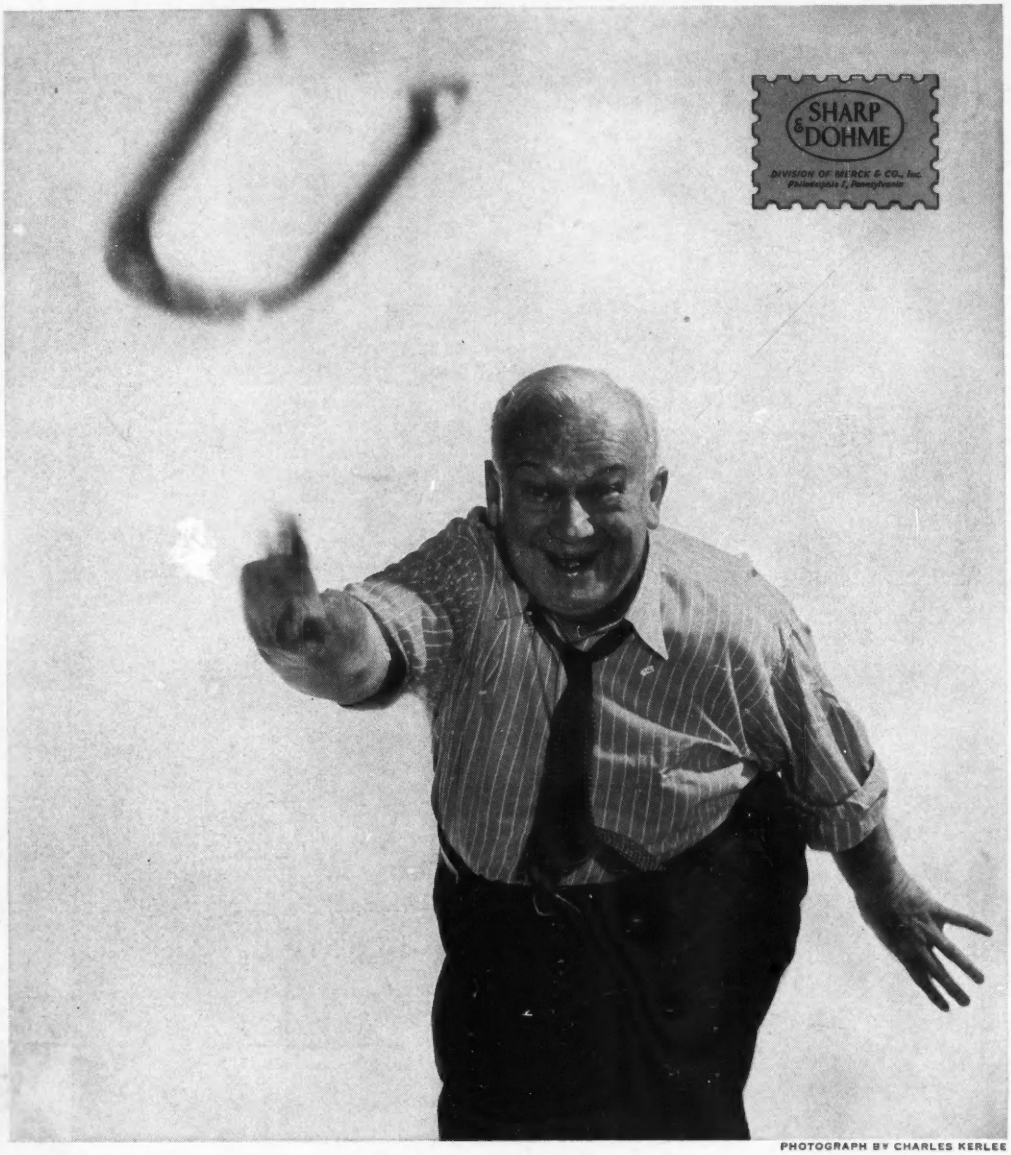
California MEDICINE

JANUARY 1955

C.M.A. Annual Meeting, May 1-4, 1955, San Francisco

OFFICIAL JOURNAL
OF THE CALIFORNIA MEDICAL ASSOCIATION

VOLUME 82 NUMBER 1



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References: 1. J.A.M.A. 153:185, 1953. **2.** N.N.R. 1953, p. 486.

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Volume 82 JANUARY 1955 Number 1

Abnormal Hemoglobins

Clinical Disorders Resulting from Various Combinations

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• Within the past few years it has been noted that abnormal types of hemoglobin found in certain persons are associated with definite clinical disorders. At least four different varieties of sickle cell anemia are now recognized, three of them being heterozygous and one homozygous. When the gene for sickling is represented once, the person is asymptomatic and is said to have "sickle cell trait." However, when the sickle cell trait is present in combination with certain other hemoglobin abnormalities such as hemoglobin C or D or with thalassemia trait, symptomatic clinical disease results. The homozygous condition, in which two genes for hemoglobin C are present in the same person, has been observed in a few instances. A similar condition as regards hemoglobin D has not as yet been recognized.

THE CLEAR-CUT DEMONSTRATION by Pauling and coworkers[6] in 1949 of the presence of hemoglobin, electrophoretically different from normal, in subjects with sickle cell trait or disease served to stimulate tremendous interest in human hemoglobin. Until then, fetal and adult hemoglobins constituted the only known varieties of human hemoglobin. Von Körber[4] in 1866 and Von Krüger[5] in 1877 established the fact that fetal hemoglobin in an alkaline medium was much more resistant to denaturation than adult hemoglobin. In 1951, Singer, Chernoff and Singer[12] reported data that established the presence of fetal hemoglobin in a number of different kinds of anemia. It was postulated that "the resistant hemoglobin fractions in these disorders represent either a continuation or a reactivation of the production of the embryonic pigment." Thus, fetal hemoglobin is not, in fact, an inherited abnormal hemoglobin. Its presence would seem to be best explained by reversion to a normal embryonic process.

However, following the demonstration of an abnormal hemoglobin associated with the sickling phenomenon, two additional hereditary abnormal types of hemoglobin were reported.[1, 2] These abnormal hemoglobins have been designated as hemoglobin C and hemoglobin D. Thus, at present there are five recognized types of hemoglobin in humans, namely, normal (A), fetal (F), sickle cell (S), C, and D. Table 1 gives the principal known facts about these various types of hemoglobin. Hemoglobins A, C and S are readily distinguished electrophoretically from one another, S occupying an intermediate position as regards mobility between A and C. On the other hand, hemoglobin D exhibits the same electropho-

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Presented before the Section on General Medicine at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

TABLE 1.—*Species of human hemoglobin (modified after Itano, H. A., Proc. Nat. Acad. Sci., 37:775, 1951)*

Name of Hemoglobin	Letter Designation	Causes Sickling	Electrophoretic Mobility*	Solubility†	Resistance to Alkali Denaturation
Normal adult	A	No	Slow	High	Normal
Normal fetal	F	No	Slow	High	Increased
Sickle cell	S	Yes	Intermediate	Low	Normal
.....	C	No	Fast	High	Normal
.....	D	No	Intermediate	High	Normal

*As carbomonoxyhemoglobin in cacodylate-sodium chloride buffer of pH 6.50 and ionic strength 0.1.

†As amorphous ferrohemoglobin in concentrated phosphate buffers of pH 6.8 at 18° C.

retic mobility as hemoglobin S and can be differentiated only by the facts that (1) its presence does not make the red cell capable of sickling as does hemoglobin S, and (2) it differs in solubility from hemoglobin S. Hemoglobin F has the same electrophoretic mobility as hemoglobin A but is distinguished from hemoglobin A by its resistance to denaturation in an alkaline medium. The hemoglobin that is present in subjects with thalassemia or the thalassemia trait has the same electrophoretic mobility as normal hemoglobin. As yet, no characteristic variation of this hemoglobin has been found to justify any statement that it is different from normal hemoglobin. It is conceivable, however, that it may be shown to be different at some future time. Indeed, the development of one of the forms of sickle cell disease on the basis of the presence in the same individual of the sickling trait and the thalassemia trait may be due to some as yet undiscovered abnormality in the hemoglobin that is found in subjects with the thalassemia gene.

The foregoing discoveries are of a great deal more than theoretical importance. The most obvious and outstanding practical clinical outgrowth has been the demonstration of the presence of four distinct types of sickle cell disease on a genetic basis. Before the recognition of abnormal adult hemoglobins, only one type of sickle cell disease was known, namely, that which occurred in certain of the offspring of parents both of whom had the sickling trait. It is now well established that sickle cell disease may result from the presence in the same individual of one sickling gene with the following:

1. Another sickling gene—that is, homozygosity for sickling.
2. The gene responsible for thalassemia.
3. The gene responsible for hemoglobin C.
4. The gene responsible for hemoglobin D.

Charts 1, 2, 3, 4 and 5 show the typical patterns of inheritance of the genes for sickling and hemoglobin C and the four genetic situations in which sickle cell disease is known to occur.

Powell, Rodarte and Neel[7] were the first to report in the medical literature of the United States a case

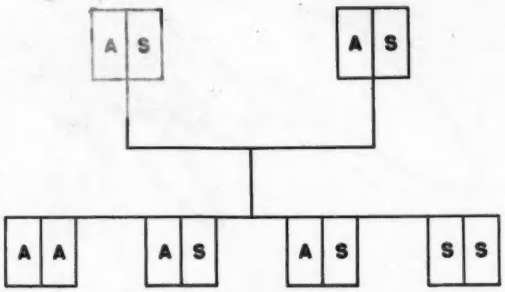

Chart 1.—Inheritance of the gene for sickling.

of sickle cell disease in a person heterozygous for sickling. The patient was shown to have inherited the sickling gene from one parent and the thalassemia gene from the other parent. While Silvestroni and Bianco[9, 10, 11] had previously reported from Italy a series of five families with this same clinical picture, the presentation of crucial evidence supporting the coexistence of the sickling and the thalassemia genes had not been made. Itano and Neel[1] made the first report of sickle cell disease due to the presence of the sickling gene and the gene for hemoglobin C. In 1951, Itano[2] first recorded data on a patient with sickle cell disease due to the presence of the sickling gene and the gene responsible for hemoglobin D.

It would appear, therefore, that the presence of the sickling gene in association with the gene for one of the other abnormal hemoglobins or with the gene responsible for thalassemia may result in sickle cell disease. Certainly this is true in the instances cited. Whether it will hold true for other abnormal hemoglobins, if such are found, cannot be stated. It can be anticipated that rapid developments in this field will occur and, in fact, during the period of preparation of this report Itano, Bergren and Sturgeon[3] obtained evidence for still another abnormal hemoglobin designated as hemoglobin E. Furthermore, many other combinations of abnormal hemoglobins can be projected. Just what such combinations will bring about clinically is unknown. The ground has hardly been broken in this new field of hematology.

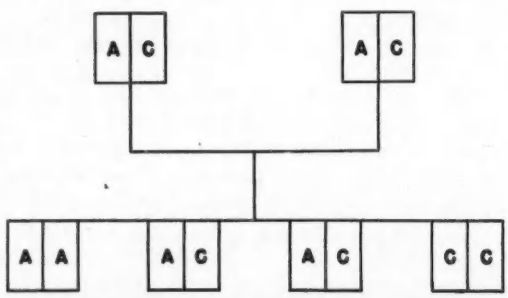

Chart 2.—Inheritance of the gene for hemoglobin C.

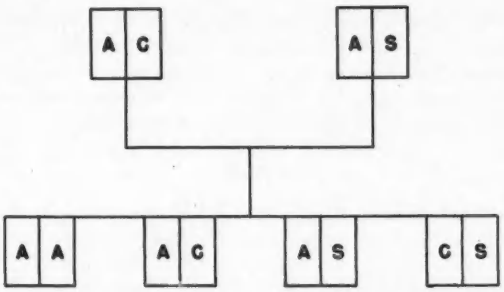

Chart 3.—Inheritance of combinations of the gene for sickling and that for hemoglobin C.

No report of the presence in the same individual of the following combinations has come to notice: CD, CT,* DD and DT.* However, a few cases in which homozygosity for C was present have been reported.[8, 13] Subjects in whom this condition exists apparently do not have severe symptoms. Stained smears of blood show large numbers of target cells and there may be mild reticulocytosis and splenomegaly.

Conditions observed by the authors in subjects with the following will be reported in brief:

1. Homozygous S, i.e., two genes for hemoglobin S.

2. Hemoglobin C and S, i.e., one gene for hemoglobin C and one gene for hemoglobin S.

3. Homozygous C, i.e., two genes for hemoglobin C.

CASE REPORTS

Homozygous Sickle Cell Disease

A 23-year-old Negro man was admitted to Wadsworth General Medical and Surgical Veterans Administration Hospital for the first time on January 29, 1954. The main complaints were shortness of breath and pain in the legs. All his life the patient had had dyspnea and had tired quickly. He had been discharged from the Army in August 1952, after a short period of service, because of fever, jaundice, leg pains, dyspnea and anemia. His parents and three of his brothers were well but one of his brothers was said to have been jaundiced for a week two years ago.

The patient was poorly developed, slender and eunuchoid in appearance with a short trunk and long, slender extremities. The fingers were suggestive of arachnodactyly. Icterus of the sclerae was noted. The precordium was hyperactive and there was a Grade II blowing systolic murmur at the apex. The heart was at the upper limit of normal size. The liver and the spleen were not palpable.

Erythrocytes numbered 2,080,000 per cu. mm. and the hemoglobin content was 6.4 gm. per 100 cc. Leukocytes numbered 12,400 per cu. mm. The volume of packed red blood cells was 17 per cent of the whole blood. About 75 per cent of the erythrocytes in a specimen of blood obtained from a finger after constriction with a rubber band showed sickling on direct smear. There were three normoblasts for each 100 leukocytes. The reticulocyte count was 3 per cent. Normoblastic and pronormoblastic hyperplasia were noted in a specimen of sternal marrow. The icterus index was 50. Serum bilirubin was 4.55 mg. per 100 cc. (direct, 2.35, and indirect, 2.20). The urinary urobilinogen was 21 mg. in 24 hours. Analysis of hemoglobin by electrophoresis revealed the homozygous S pattern. Jaundice and the other symptoms promptly subsided after the patient was admitted to hospital.

Comment: In this case there were the usual clinical findings of moderately severe sickle cell disease. The history indicated that the patient had been subject to hemolytic crises, and admission to the hospital was due to such a crisis. Present were: Anemia, sickling of large numbers of the erythrocytes, nucleated erythrocytes in peripheral blood, mild reticulocytosis, jaundice, increased urinary urobilinogen and the homozygous S pattern of hemoglobin electrophoretically.

Sickle Cell-Hemoglobin C Disease

A Negro patient was admitted to hospital in January 1952 owing to severe abdominal pain following

* "T" is employed here to designate the gene for thalassemia although hemoglobin abnormality has not been demonstrated in the condition by methods now available.

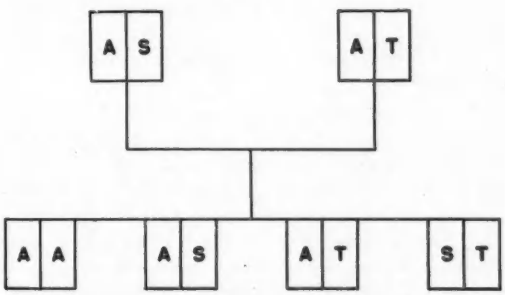

Chart 4.—Inheritance of combinations of the gene for sickling and that for thalassemia.

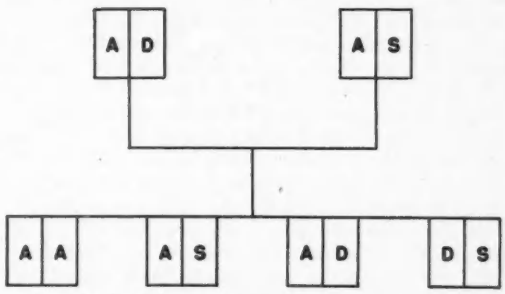

Chart 5.—Inheritance of combinations of the gene for sickling and that for hemoglobin D.

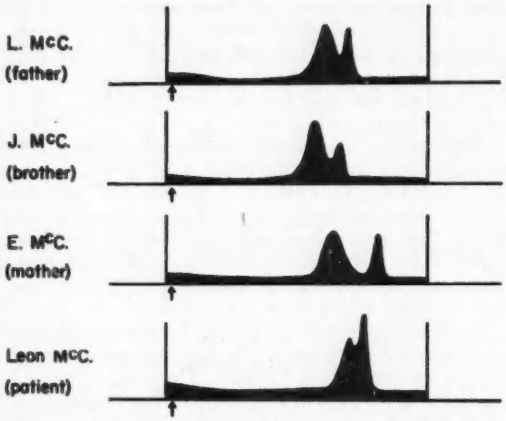

Chart 6.—Electrophoretic patterns of hemoglobin in a family with sickle cell trait and the hemoglobin C abnormality.

an epileptic convulsion. The spleen was large and an intracapsular rupture was present. No symptoms with reference to any hematological disorder were noted. Routine studies of the blood revealed no anemia, but many target cells were present. A sickling preparation was positive for sickling. Electrophoretic studies revealed approximately 50 per cent each of sickle cell and C hemoglobin. Electrophoretic study of blood from the parents of the patient demonstrated heterozygosity for sickling (sickle cell trait) in one parent and heterozygosity for hemoglobin C in the other. Chart 6 shows the electrophoretic patterns in the patient and members of his family.

Comment: Outstanding in this case were the race of the patient (Negro), splenomegaly, target cells, sickling and the electrophoretic findings of S-C hemoglobins. It is probably significant that clinically this is a much more benign form of sickle cell disease than that ordinarily found with the homozygous S disorder. Splenomegaly has been found in most of the patients with this disorder.

Homozygous Hemoglobin C

A well-nourished and well-developed Negro boy 3 years of age was examined by a physician because he was not eating well. No physical abnormalities were noted. Results of routine studies of the blood were normal except for the presence of a large number of target cells and an occasional nucleated erythrocyte. Sickling was not demonstrated. The electrophoretic pattern showed that the hemoglobin was almost entirely of the C type. Both parents showed the A-C hemoglobin pattern electrophoretically (heterozygous hemoglobin C).

Comment: The pertinent findings in this patient were the target cells, the presence of nucleated erythrocytes in the peripheral blood, the demonstration of hemoglobin C as the main type of hemoglobin in the blood cells and the absence of significant clinical symptoms. The absence of splenomegaly is probably unusual as there is some evidence that enlargement of the spleen may be found in patients with this disorder. It is entirely possible that splenomegaly will develop in later life.

DISCUSSION

That certain clinical conditions are associated with the various possible combinations of abnormal hemoglobins is just beginning to be recognized. It seems that homozygous sickle cell disease will continue to represent the most severe type of sickle cell disease although sickle cell-thalassemia disease may run a very similar course. The heterozygous S-C sickle cell disease has been studied in a number of instances and seems to be distinctly milder, as was true in one of the cases (Case 2) reported herein. Target cells are present in great abundance and the spleen is enlarged in the majority of instances. Anemia is less severe than in the homozygous S type of sickle cell disease. As only one case of the S-D type has been reported, it is impossible to make any pertinent comments on this type.

The homozygous C condition has been studied in only a few instances. Observations of the subjects indicate that abnormalities insofar as the clinical symptoms and signs are concerned are not pronounced.

If physicians were on the alert for conditions in which abnormal hemoglobins are present, probably many more clinical instances of such disorders would be noted. A few points with reference to detection of these conditions seem worthy of comment. Most of the cases reported up to the present were in Negroes and persons of Mediterranean extraction. Unexplained anemia in such persons should always raise the question of one of these disorders. If, in addition to anemia, target cells are present in appreciable numbers, suspicion should be increased. Nucleated erythrocytes, jaundice and reticulocytosis are strong suggestive findings. While the demonstration of sickling will not determine what type of sickle cell disease is present, it clearly indicates that at least one gene for sickling is present. Patients who have all or some of the findings mentioned should have detailed chemical and/or electrophoretic studies.

A concerted effort in this regard no doubt will result in the uncovering of many more cases of the already recognized clinical disorders and probably will result in the discovery of previously unidentified disorders due to other genetic combinations involving different types of abnormal hemoglobins.

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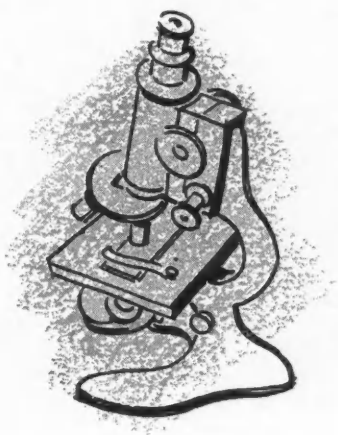

Normal Micturition

Certain Details as Shown by Serial Cystograms

FRANK HINMAN, JR., *M.D.*, GERALD M. MILLER, *M.D.*, ELDON NICKEL, *M.D.*, HOWARD L. STEINBACH, *M.D.*, and EARL R. MILLER, *M.D.*, San Francisco

• Serial cystograms were made during micturition, using a roll film cassette. It was observed that detrusor contraction can be the first activity in normal urination, immediately followed by relaxation of the perineum. The "external sphincter" can shut off the urinary stream as an isolated movement preceding elevation of the bladder base.

THE SEQUENCE in normal micturition was restudied recently by Muellner[2] by fluoroscopic examination during voiding of a radiopaque solution. He reported that increased intra-abdominal pressure and relaxation of the perineum start the voiding reflex, with the detrusor then reflexly contracting and emptying the bladder. He saw no contraction of the "external sphincter" as the initial action in stoppage of the stream.

PRESENT STUDIES OF NORMAL MICTURITION

In the present studies, on serial films using a roll film cassette (Fairchild) (Figure 1), the sequence of activities during micturition in normal women was recorded. The intervals between exposures was 0.5 seconds and the time of exposure 0.6 seconds, for a total interval of 1.1 seconds.

The relaxed bladder empties in a different fashion from that of the already contracted bladder. That is to say, a subject who has no desire to urinate has a different voiding sequence from one who has an intense urge to void. The sequence with a relaxed bladder was seen to be first contraction of the detrusor, followed immediately by lowering of the bladder base. Opening of the internal, then the "external sphincter" followed.

The contracted bladder, producing the urge to void, was seen to have an already open internal sphincter. Upon the command to void, the subject lowered the bladder base (Figure 2) and then opened the external sphincter (Figure 3).

Cessation of urination in mid-stream was brought about by voluntary closure of the external sphincter (Figure 4) followed by elevation of the pelvic musculature (Figure 5) with relaxation of the detrusor (Figure 6).

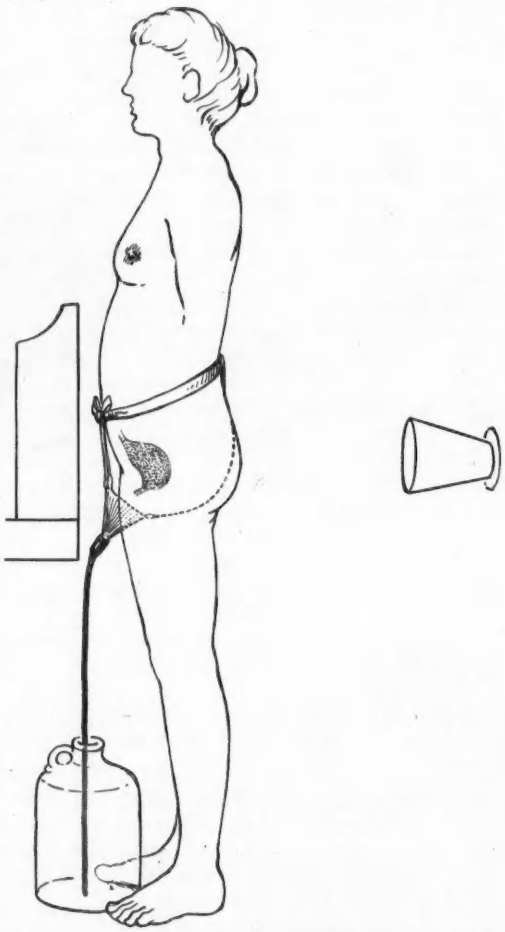

Figure 1.—Diagram showing female patient standing before the Fairchild roll film cassette. Between her legs is strapped a plastic funnel leading to a collection bottle. The bladder has been filled with 200 cc. of radiopaque solution.

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Presented before the Section on Urology at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

Some of the observations in the present study were at variance with those previously reported by other investigators. It was not confirmed that increasing intra-abdominal pressure with relaxation of the perineum necessarily initiates the voiding reflex; detrusor contraction can come first. And urination can be stopped by isolated contraction of the distal portion of the urethra (the "external sphincter" of the female). The observations herein more closely agree with those from the classic pressure recordings made by Denny-Brown and Robertson[1] 20 years ago.

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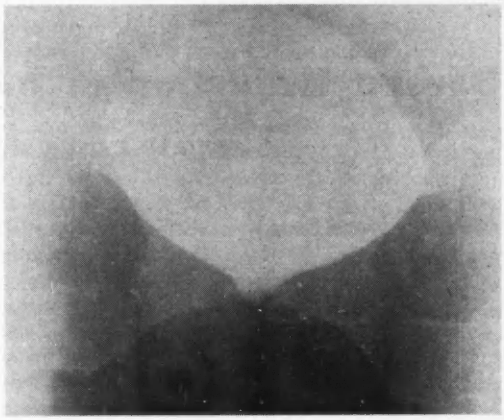

Figure 4.—Voluntary closure of external sphincter.

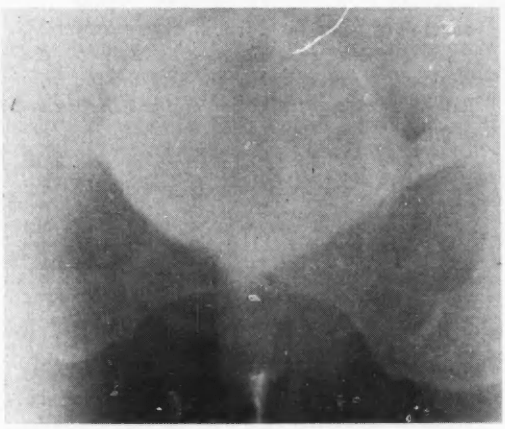

Figure 2.—Relaxation of pelvic musculature.

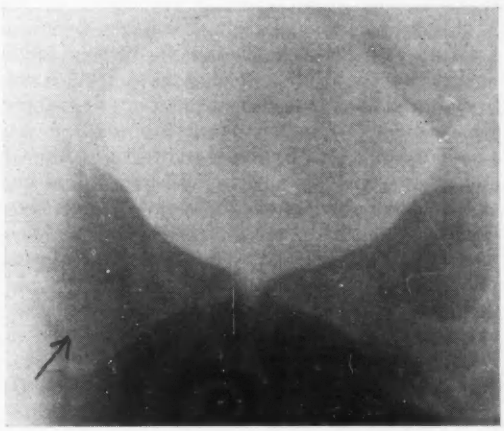

Figure 5.—Elevation of pelvic musculature.

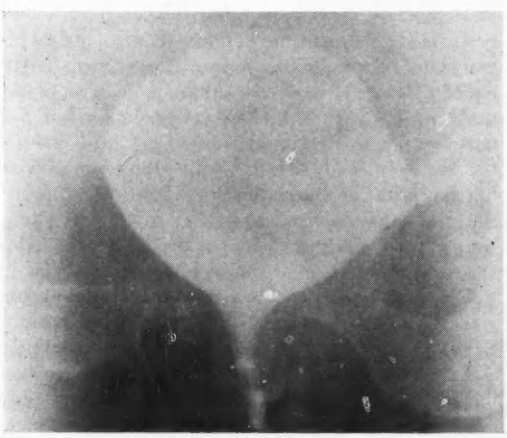

Figure 3.—External sphincter opened.

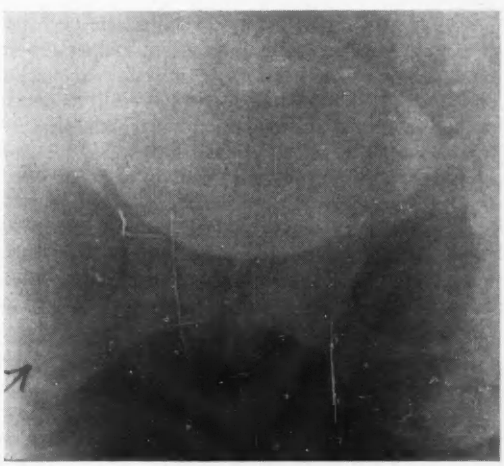

Figure 6.—Elevation of pelvic musculature with relaxation of the detrusor.

Barium Reduction of Intussusception in Infancy

EDWARD J. DENENHOLZ, M.D., and GEORGE. S. FEHER, M.D., Modesto

• Barium enema reduction was used as the initial routine treatment in 29 infants with intussusception. In 22 of them the intussusception was reduced by this means. In three of eight patients operated upon the intussusception was found to be reduced. Four of the remaining five patients had clinical or x-ray evidence of complications before reduction by barium enema was attempted.

Twenty-one of the patients, all of whom were observed in private practice, were treated without admission to the hospital. After reduction, these patients were observed closely by the clinician. None of these patients showed clinical or x-ray signs of complications before reduction.

Certain clinical and roentgen criteria must be satisfied before it can be concluded that reduction by barium enema is complete.

If there are clinical signs of complications with x-ray evidence of small bowel obstruction, only a very cautious attempt at hydrostatic reduction should be made. As the time factor is generally a reliable clinical guide to reducibility, the late cases should be viewed with greater caution. Long duration of symptoms, however, is not per se a contraindication to an attempt at hydrostatic reduction.

OPINION AS TO the relative merits of the operative and nonoperative treatment of intussusception in infancy varies widely. One view, probably that of a majority of surgeons and pediatricians, is that this condition demands prompt surgical intervention and that barium enema has no real place in the therapeutic regimen. Ravitch[16] and others,[4] however, believe that treatment of an infant with intussusception should be begun with an attempt at hydrostatic reduction, and that the majority of patients with intussusception in infancy can be successfully treated by this means. Although this nonoperative treatment has long been the method of choice in the Scandinavian countries,[11, 12] and in Australia,[6, 10] it has been looked upon with some distrust in this country. Renewed interest in the nonsurgical method was stimulated, however, by the observations of Ravitch and co-workers.[14, 15, 16] After extensive clinical and experimental observation, these investigators concluded that mortality, morbidity, and length of hospital stay are all lessened by the hydrostatic as compared with the surgical method. Ranging between these two opposing beliefs are numerous gradations of opinion, including the proposal that barium reduction may be employed, but that it should be followed in each instance by laparotomy to verify reduction.

PRESENT STUDY

The present report is based on observation of 29 consecutive infants with intussusception who were treated in private practice in a four-year period ended in April 1954. The infants in this study ranged in age from 5 months to 32 months. Fifteen were under one year of age, 10 between one and two years, and four over two years of age. The duration of symptoms at the time the patient was first observed varied from one hour to five days. In eight cases symptoms were present for less than 12 hours, in ten from 12 to 24 hours, in three from 24 to 48 hours, in three from 48 to 72 hours, and in five over 72 hours.

All infants suspected of intussusception were given a barium enema examination to confirm the diagnosis; and, if intussusception was observed, hydrostatic reduction with barium was used as the initial treatment. All the barium reduction procedures were performed by one of the authors (G.S.F.) or by his associate. The clinical examinations were made either primarily, or in consultation, by one of the authors (E.J.D.) or his associates, in all but five patients. These five patients were referred directly for roentgen study by the family physician. With the exception of one patient in whom the examination was made in the hospital, all x-ray procedures were carried out in the office of the radiologist. This was originally done because of the superior facilities in the office as compared with the hospital, but was continued because of the gratifying results. Early in the study, a surgeon was notified routinely that barium reduction was being attempted, so that the operating room could be alerted. As the study progressed, this procedure was followed only in complicated cases in which it was suspected barium reduction might not be effective or feasible.

Presented before the Section on Radiology at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

Patients in whom barium reduction was successful were not admitted to the hospital. Instead, they were observed in the radiologist's office for 30 minutes to one hour, and if no evidence of recurrence of symptoms developed, the patient was permitted to return home. In the majority of cases, the pediatrician was present for all or part of the procedure and instructions to parents as to care of the patient at home were given by him. Fluids were given by mouth as soon as the infant appeared to want them after reduction. Antibiotics were administered to some of the infants in whom the intussusception had been present for over 24 hours as a prophylaxis against specific diarrhea, and antibiotic therapy was also continued in cases in which they were already being administered for antecedent illness. The parents were asked to report immediately any recurrence of symptoms and to make a progress report by phone in two to eight hours, the time of report depending on the time of day or night and upon the nature of the case. They were informed of the probable development of loose stools for a day or two and of the possible presence of blood and mucus in the first stools following reduction. In all cases it was asked that the patient be returned to the pediatrician the following morning for examination even in the absence of symptoms. The one patient in whom the intussusception was reduced in the hospital (at 2 a.m.) was discharged eight hours following the reduction. The patients in whom reduction was considered not to have been effected were immediately referred to the hospital. In all but two of these patients the condition was recognized as late or complicated, and the probability of the need for operation had already been discussed with the referring family physician or with the surgeon when the nature of the case was revealed to the pediatrician or the radiologist.

TECHNIQUE OF BARIUM REDUCTION

The technique of hydrostatic reduction has been described in detail by Ravitch and McCune,[16] and by Girdany, Bass and Grier.[4] Since the authors have employed some modifications, however, a brief outline may be worth while.

The examination begins with a plain film of the abdomen. If this shows evidence of small bowel obstruction and/or peritonitis, barium enema is performed with great caution.[7] (This policy was adopted because in earlier studies it was observed that in cases in which there was clinical evidence of complications, such as peritonitis, intestinal obstruction, toxemia and prostration, and x-ray evidence of small bowel obstruction, hydrostatic reduction was not successful.) Before the x-ray studies are started, the parents of the infant are instructed in immobilizing the infant, and particularly in the manner of compressing the baby's buttocks to prevent expulsion of the tube. Enlisting the aid of the parents serves two purposes: (1) it is psychologically sound, since the parents feel that they are actively aiding in the treatment of their child, and it avoids anxieties that may result when they are not permitted in the examining room or when they view formidable restraints. (2) It prevents undue exposure of the x-ray personnel. No mechanical restraints or sedation were employed.

A child's-sized, ungreased Bardex catheter is inserted into the rectum and inflated. The barium is permitted to enter the rectum and distal colon. After the intussusception has been recognized fluoroscopically, spot films are made. Reduction is then begun by slowly elevating the enema container from a position slightly above table level to that needed to overcome the resistance of the intussuscepted area. In the majority of cases this is three to three and a half feet above the level of the table. In no case is the container raised more than four feet above the top of the table. During the reduction, spot films of the advancing barium column are made. In cases in which there are no complications, if the barium reaches an impasse and does not enter the terminal ileum within a few minutes, the patient is allowed to evacuate and the procedure is begun again. In some cases reflux into the ileum occurs during the evacuation, and the intussusception is found to be reduced. At times evacuation is accomplished accidentally and after the second filling the reduction is then completed with relative ease. No more than three fillings are used in any patient, and rarely is it necessary to use even three. At no time is manual palpation used. In the late or complicated cases, as evidenced by clinical and x-ray findings, only one cautious attempt at barium reduction is made. If this is unsuccessful, the patient is promptly referred for operation.

CRITERIA FOR REDUCTION

In the present series certain clinical and radiologic criteria had to be satisfied before reduction was considered complete. Clinically, a dramatic and striking change in the patient is evident when reduction has been effected. An infant who previously has been screaming in intense agony, or who has been extremely apathetic and listless between episodes of abdominal pain, will abruptly fall into a deep sleep on the examining table, or will begin to smile or become active or engage in play. If an abdominal mass was previously noted, it will have disappeared.

Many patients, especially if treated early, will show no particular symptoms during the 24 hours following reduction. It should be recognized that some patients will have loose stools (postreduction diarrhea) for one to two days following reduction. Blood in the stools with mucus is also often present

the day following reduction. Occasional abdominal pain or cramp, usually not severe, may less frequently occur. These symptoms are similar to those that are seen after surgical reduction, but are generally much less severe. Observation of the patient soon reveals that reduction has been complete, since the pain is only fleeting, the infant is active, stools without blood are passed, and vomiting, distention, shock, and abnormal abdominal findings are not present. It is of utmost importance to recognize the postreduction symptoms for what they are, for if misunderstood they may lead to considerable anxiety and even unnecessary operation. Fever, except of slight degree, is not usually present after barium reduction, as it is after operative treatment.

Radiologically, reduction is diagnosed when the cecum is filled and there is a free flow of barium into the terminal ileum. It is important to recognize that a filling defect at the ileocecal valve, after the terminal ileum has been filled, is often due to edema of the mucosa, rather than to incomplete reduction.

RESULTS

In 22 of the 29 patients reduction was successfully accomplished by means of barium enema as described.* In all patients in whom intussusception was present for 48 hours or less, barium reduction was successful. One of these patients was operated upon, however, because of the family physician's insistence, and at operation the intussusception was observed to be reduced. In two additional patients early in the series the radiologist was not certain that complete reduction had occurred and operation was carried out. In both instances, the intussusception was found to be reduced. It is recognized that anesthesia may have brought about the reduction in these patients, and they are not considered as having had reduction by hydrostatic means. In the remaining five patients, barium reduction was unsuccessful and operation was performed. These patients first came under observation late and the duration of symptoms before barium reduction was attempted was 50, 74, 88, 96 and 120 hours. In one of these patients the intussusception was found to be partially reduced immediately distal to the cecum and was easily reduced at operation. In four patients clinical evidence of complications, such as toxemia, prostration, fever (102° to 104° F.) and a variable degree of distention, was present. In three of these patients there was evidence of small bowel obstruction on a plain film of the abdomen. In all five patients intussusception of considerable magnitude was observed at operation. The intussusception was reducible at operation in three patients and in the other two it was considered nonviable and irreducible. In one of these a primary resection was done, with recovery. Two patients died. One of them (who had had symptoms for 96 hours before operation) died of toxemia about 40 hours after operation. The other (in whom symptoms had been present for five days) died 12 hours postoperatively of peritonitis and toxemia.

*Since this report was written, intussusception was reduced by barium enema in three additional early cases.

DISCUSSION

The obvious advantage of barium reduction is that in many instances the infant is spared anesthesia and an abdominal operation, although the advocates of operative treatment point out that the surgical mortality is low. A comparison of mortality rates by the two methods of treatment has been cited by others. Our small series does not lend itself to such a comparison. Another advantage is the lessening of morbidity. The studies of Ravitch,[15, 16] and of Bass and Girdany,[1] indicated a considerable shortening of the hospital stay as well as a decreased incidence of fever, diarrhea and vomiting in the nonoperative patients as compared with the operative.[1] As far as is known, the present study is the only one in which the majority of the patients were treated on an outpatient basis. Of 28 patients reported by Bass and Girdany,[1] four were returned home immediately after operation. Twenty-one of the patients in the present series were not admitted to the hospital at any time. It should be pointed out, however, that all these patients were observed in private practice, that they could be followed closely, and that parental cooperation could be expected. It should be emphasized that this procedure would not be applicable, except in selected instances, in clinic practice.

As was stated previously, the major objections that have been raised to reduction of intussusception by this method are that: (a) gangrenous bowel may be reduced; (b) a rupture of the bowel may be brought on by the barium enema; (c) a specific etiologic factor for the intussusception, such as a Meckel's diverticulum or polyp, may be overlooked; (d) ileoileal intussusceptions cannot be diagnosed by this method; (e) it is difficult to be certain by x-ray that reduction has occurred; and (f) the procedure is tedious and may thus cause serious delay in the patient's treatment. Experimental studies have shown that gangrenous bowel cannot be reduced by this method, and that much greater pressures than those used for reduction are required to produce intestinal rupture.[11, 15] The burden of the pressure in the hydrostatic method is borne by healthy bowel and is diffusely distributed. Actually, the method is quite similar to that employed for reduction at operation. Essentially, the surgeon by squeezing the bowel distal to the intussusception increases the intraluminal pressure and forces the intussusception back. This is precisely what the barium

does in the hydrostatic pressure method, except that the enema does not contuse the bowel wall or abrade the serosa.

In the majority of instances of intussusception in infancy no specific etiologic factor is found at operation. Lesions such as Meckel's diverticulum or polyps are more likely to be encountered in older children. In no instance in this series did a patient return because of symptoms that could be attributed to a causative lesion that was missed at operation. There were no recurrences in patients in the present series. The authors agree with Ravitch and Morgan[16] that, should recurrence develop, barium reduction should be done and the abdomen then explored for the presence of a possible causative lesion.

In the great majority of cases, intussusception is ileocecal or ileocolic. There were no cases of ileoileal intussusception in the present series. This condition cannot be diagnosed with certainty by the method described herein, but in the event of failure to find barium enema evidence of ileocolic intussusception, in the face of a suggestive clinical picture, together with x-ray evidence of small bowel obstruction, the authors would promptly advise surgical intervention.

The diagnosis of adequate reduction can usually be readily made by means of the barium enema. One of the troublesome features is the presence of a filling defect at the ileocecal valve following attempt at reduction. In the authors' experience, if the barium flows freely into the terminal ileum and if the patient shows obvious improvement as previously noted, the defect is due to edema and does not indicate incomplete reduction. The identical defect has been observed in patients who were given barium orally after successful surgical reduction.[4] If the criteria previously cited are not present, the patient should be promptly subjected to operation.

Ileocecal or ileocolic intussusception can readily be diagnosed, even before clinical symptoms are acute, by means of barium enema. Although many observers[2, 5, 13] feel that barium enema is rarely needed to confirm the diagnosis, the authors have found it a very helpful clinical tool. Since the patients in the present series were observed in private practice, they were, as a group, brought to us for examination earlier than would patients in a clinic series. Five of the patients were seen between one and four hours after the onset of symptoms. It is in early cases that the barium enema is of most value. Perhaps the infant's color is good and he plays between attacks. There may be no shock, no bloody stools or bloody mucus on the examiner's fingers and no abdominal mass present. A plain film of the abdomen may show no abnormality. Oftentimes the infants have either mild enteritis or upper respiratory tract infection preceding the attack, and these conditions may cause deceptive variations in the clinical picture. It is in such circumstances that barium enema may be most helpful either in confirming the diagnosis or in showing a free flow of barium into the ileum. And in either event it is probable that the patient can be spared operation. If intussusception is present, an attempt at barium reduction may be made readily. It is possible that the feeling which appears to exist among some surgeons that intussusception is easy to diagnose, may well have resulted from the fact that the patients they examine are often first screened by the pediatrician or family physician.

The procedure need not usually be tedious or time-consuming. It can be started much sooner than an operation and it takes about 20 to 30 minutes. If operation has to be done later, in some instances, the intussusception will have been partially reduced by the enema, thus facilitating the surgical procedure. Ravitch[16] advocated a simple McBurney incision in such instances.

The time factor is of great importance in the success of barium reduction. Generally speaking, the earlier the patient is treated, the more readily can barium enema reduction be effected. Irreducibility is determined by adhesions between the sheaths and by the degree of edema. Both factors increase with time. Duration of symptoms alone is not entirely reliable however; in a few late cases in the present series, reduction was relatively easy and in a few early cases it was more difficult. Although not observed by us it is conceivable that complications might be present in early cases. In cases in which there were complications, intestinal obstruction or peritonitis, reduction was not accomplished with barium enema; but in those cases it was done only with difficulty at operation, if it could be done at all.

In the present series an attempt was made to obtain close cooperation between the pediatrician and radiologist. The responsibility for prompt referral of the patient to the radiologist for an attempt at reduction and the close observation and contact with the patient following reduction rested primarily with the clinician. The radiologist's responsibility lay in adequately carrying out the attempt at reduction and in the x-ray recognition of a successful reduction or of failure. The decision to desist or persist in efforts at reduction was a joint one, arrived at in each case by an appraisal of the entire clinical picture. The great majority of the reports in the literature concerning the treatment of intussusception and evaluating the various methods of treatment have been made by surgeons. Ravitch, a surgeon and proponent of barium reduction, emphasized strongly that barium reduction is a surgical procedure. The authors feel that it is primarily a roentgen technique to be pursued by radiologists in close cooperation with pediatricians, general physicians or surgeons, as the situation presents. It is of interest that of the

patients seen by the authors from the outset of symptoms, there was only one in the entire series subjected to operation. In that instance, early in the series, the clinician and the radiologist felt some doubt that successful reduction had occurred; but at operation complete reduction with viable bowel was observed.

We concur with Girdany, Bass, and Grier that the radiologist should become fully acquainted with the clinical aspects of intussusception and expert in the technique of hydrostatic reduction. Interest and willingness are most important; half-hearted, routinized attempts at reduction are usually doomed to failure.

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Instruction of Mothers in Well Baby Care

A Program of "Talks" at Monthly Visits

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• *As advisor and counsellor of mothers, a physician can contribute greatly to preventive medical care of infants. Advice can be given piecemeal, but according to a program.*

At birth the mother is reminded of the importance of emotional environment in shaping the life and personality of the child. The most important thing the mother and father do during the first year is enjoy their baby.

At six months each monthly visit includes a discussion period. First is the concept of balanced diet and control of diet. At the seventh month, the mother learns how to report an illness—how to get information she wants and to give information the physician wants. Eighth month: Care of minor illness—necessity for rest and the importance of withholding food to promote rest; and the importance of fluids. Ninth month: Discussion of shoes. Eleventh month: Training for toilet and other habits; discussion of attitude toward thumb-sucking. Twelfth month: Stress household accidents, which cause more deaths than all infectious diseases combined.

AS ADVISOR AND COUNSELLOR of mothers of infants in matters of preventive medical care of their babies, the author follows a procedure that is a means of giving a short course of infant care to every new mother along with the monthly supervision of her infant, especially through the second six months.

Soon after the birth of an infant the mother is reminded of the importance of emotional environment in shaping the life and personality of the child. Particular emphasis is placed on the mother and father enjoying their baby during its first year, since the direction of the child's disposition and personality is largely determined by the time he is one year old. The pattern may not be fixed at this time but will become so unless changed.

The parents must not be afraid to enjoy their baby and they should not confuse enjoyment with spoiling. Spoiling the child is teaching him to be unpleasant to get his normal needs. Bakwin[1] put it this way: "If a child feels confident of his parents' affection, esteem and respect, the parents can do almost everything wrong and the child will still turn out all right."

A great help in the enjoyment of the baby is the simplification of feeding brought about in recent years by modern methods of sanitation and preparation of milk and baby foods. One of the great advantages of breast feeding is that it simplifies care and, in freeing the mother from tedious time-consuming tasks, allows her time to enjoy the baby.

A new mother will be flooded with good advice freely bestowed by friends and relatives. She will need a place to park these well-meant but superfluous gems. She may tactfully say, "I will make note of your suggestion to ask the doctor." Thus, she removes the worry from her mind and flatters the would-be benefactor. A "worry list" saves the mother a lot of anxiety.

During the first six months concern is largely with diet and formula changes and additions. Modern sanitation, pasteurization, and almost universal home refrigeration, as well as the many excellent prepared evaporated milk and ready-made powdered preparations, have made formula problems largely a thing of the past. In recent years various formulas have been advocated as ideal—high protein, high calorie, plain whole milk, skim milk, pasteurized milk—all with equally satisfactory results. The simple procedure of adding a sufficient amount of boiled water to a can of evaporated milk to make one quart of formula makes a very simple and satisfactory feeding. Later, adding cereal for carbohydrate when needed avoids the conditioning of a baby to sweets.

The rush to start semi-solid foods motivated by well meaning and eager food companies is subsiding, for better reasoning. It is being recognized that many foods are not well assimilated during the first three months. Foods need be added only when they fulfill a purpose—not by age. Seeing pureed food so readily recognized in the stool the next day makes one wonder if it would not save the baby a lot of trouble to put the stuff in the diaper in the first place.

The canned vegetables, fruits and meats are great labor savers providing they are not set up as little tin gods—destroying mother's confidence in her own cooking. Mother may be afraid to feed the baby anything she has cooked herself. Unfortunate is the

Presented before a Joint Meeting of the Sections on General Practice, Pediatrics, and Obstetrical Gynecology at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

family that raises a baby in its home without improving the whole family's diet.

The modern trend in immunization has moved to a younger age. About three months seems to be the average now for starting. This is the time for maximum protection against whooping cough and immunization causes the infant less emotional disturbance. The immunization schedule shown here is one used by many pediatricians.

3 months	DPT*		Smallpox
4 months	DPT		
5 months	DPT		
1 year	DPT	Tuberculin	
3 years	DPT	Tuberculin	
5 years	DPT	Tuberculin	Smallpox
7 years	DPT	Tuberculin	
10 years	DPT	Tuberculin	Smallpox

*Diphtheria-Pertussis-Tetanus.

During the second six months, each monthly visit has a discussion period in which attempt is made to highlight some important concept of the total picture of child care. At this time attention is given to "a balanced diet":

Milk	1 pint, minimum; 1 quart, maximum
Protein	1 egg or 2 tablespoons of meat or fish
Vegetable	2 to 5 tablespoons
Fruit	2 to 5 tablespoons
Cereal	2 to 5 tablespoons
Vitamins	
Sun Baths	

Attempt to get the parent to see an overall picture of a whole day's diet rather than two tablespoons of cereal at 10 a.m., etc. It really doesn't matter which food is given at any one meal.

A balanced diet can be based on the type of food least well taken; then equivalent proportions of all other foods may be given. It would be impossible to make the child take more of what he likes least. The old insistence that "a child should clean up his plate" should give way to its original meaning that not so much should be put on the plate. It could not possibly mean, as is often interpreted, that a child should eat all that someone puts on his plate.

Every child is born with certain inalienable rights which he defends to his utmost. These rights are: to eat, sleep, breathe and go to the toilet without interference or regimentation. Even a gentle dog growls when someone comes near him while he is feeding.

At the seventh month the mother is instructed on how to report an illness. She is taught how to collect and organize the information a doctor will want, specifically: (1) name and age; (2) main complaint; (3) symptoms and temperature, in order of occurrence; (4) treatment so far. She is told, also, to have paper and pencil ready at the time she calls. This little outline was conceived for the sole purpose of saving the physician endless listening and sifting of irrelevant data. In practice, this outline has halved the time consumed by numerous phone calls.

At the eighth month, instruction on the subject of the care of minor illnesses is given according to this outline:

I. *Rest*	II. *Fluids* (water)
Bed	Elimination:
Light diet	Bowels
Aspirin	Urine
Sedatives	Sweat
Nose drops	Breath
Cough syrup	Mobilize defenses
Steam	Regulate body chemistry
	Control temperature

In the older child, rest means in bed. Usually, sometime before the child is two years old, there comes an illness that is the golden opportunity to teach the child that when he is ill he stays in bed. This saves endless vacillation later on.

The most fruitful means of promoting rest lies in a light or reduced diet. Nature is not mistaken when she takes away the appetite at the first sign of illness. It takes more energy to digest the food eaten in one day than one would possibly exert in physical or muscular effort in a day. Appetite is an accurate barometer of well-being but it is as irrational to try to make a child well by making him eat as it is to heal a broken leg by making him walk on it.

The medical profession owes an explanation as to why physicians so glibly prescribe aspirin for children and then are astonished to see adults eat it like popcorn. The difference can be simply stated by saying that we put a child to bed and give him aspirin while an adult takes aspirin instead of going to bed. The chief value of aspirin for children is to relieve the aches and pains that make it difficult for him to rest. Its role in reducing body temperature has been over-rated. Sedatives, nose drops, cough syrup and steam inhalations, when they do any good, do it mainly by promoting rest and ease.

When instructions are given about fluids, the word means, primarily, water. Mothers have to be reminded that juices are mainly to provide taste. The primary purpose of fluids is to promote elimination. In medicine a generation ago castor oil and epsom salts performed this function, but now it is known that urging or forcing of fluids does it better with less side effect. There is an old medical saying that there never was a constipated water-drinker. This is not the whole answer but it emphasizes a basic truth. Scant or dark urine is a warning of dehydration. Even the sweat glands are important in elimination. That it takes fluids to breathe is seldom thought about. Especially when the nose is blocked the fluid loss through the breath can exceed that excreted by the kidneys and the breath is an exceedingly important means of elimination of noxious products.

All the defense mechanisms of the body require

fluids to mobilize the antibodies from their sources, to transport them and insure their ultimate function. Seventy per cent of the body weight is water. Of this 50 per cent is in the cells, 5 per cent in the blood stream. Only the 15 per cent intercellular fluid can be utilized to make up deficiencies and this is the site of the immunity response.

Body temperature can range from the eighties to 105° Fahrenheit without serious consequence but the pH of the blood must remain within a range of one point to sustain life. Chemical imbalance seldom occurs when sufficient fluids are available. That the human animal is dependent on fluids for temperature control is well known.

Now when the importance of fluids and rest is explained to a mother, she will not say, "What can I do for my child?" These fundamental symptomatic treatments rank in importance with specific therapy for more serious illnesses.

When the child is nine months of age, the use and misuse of drugs is discussed with the mother. Few households have adequate antiseptics. The purpose of antiseptics is to kill bacteria. It seems reasonable that every family with children should have available solution merthiolate for fresh cuts, tincture of mercresin (stainless) for infected lesions and, perhap, antiseptic soap.

It can be explained that antibiotics do not kill but only inhibit bacteria, as the name suggests. A homely simile that seems to carry this impression is that you or I could lick our weight in wildcats if someone would tie them down but we don't want them untied when we have just started. Thus with antibiotics it is desirable to give adequate doses, at the outset, and to continue them long enough. It is true that in adults, who have a ready immune response, a single dose of penicillin at the onset of an infection can inhibit the invasion long enough for the body to overcome the infection. But in a child the same process is like damming back a flood of water—then releasing it to deluge him before his defenses are up.

At ten months, in discussing shoes, it is pointed out that there are only three reasons for wearing shoes: appearance, warmth, and protection from the hard surfaces upon which we live. Ordinary shoes do not support either the foot or the ankle. Babies' legs, at nine months to one year, look bowed because of under-developed anterior tibial muscles which support the arch. At this age a child is constantly kicking his toes for sheer glee and in so doing duplicates exactly all the tedious exercises that physical therapists prescribe for flat feet. A rigid shoe, at this age, is a most effective inhibitor of development of the arch.

An orthopedic or corrective shoe may give support but a normal shoe is an apparatus to hold a sole on the foot. A thick sole is useful for absorbing the shock of pattering feet on hard surfaces.

At the eleventh month some of the habits of infants are considered, such as toilet training and thumb-sucking. A toilet is a comfort station, not a duty chair. A child's experience on the toilet should be a relief. He must be relaxed to get relief. Force or regimentation will not relax him. It is dangerous to set an age because there is no uniform time at which a child is ready for this relief.

Thumb-sucking is a problem that has confused and frightened many mothers. A certain amount is not abnormal and not, generally, harmful. But sometimes advice to let it alone forces inhibitions and fear upon a mother. She needs release to handle the problem as seems fitting to her. She should not be afraid either to let the baby suck or to stop him, as she wishes. The concept that he must suck for some inner security or satisfaction has, at times, been over-emphasized—so much so that the mother may actually feel duty bound to encourage him to seek this perverted sense of satisfaction. Not interfering with the child who wants to suck does not mean that he must be kept sucking for fear he won't get enough.

At the end of the first year is the opportune time to discuss one of the most important parts of child care—accidents. Accidents cause more deaths of children than all infectious diseases combined. As the physician completes the immunization procedures by giving the first booster and applying the tuberculin test, he can call attention to some of the most common causes of accidents: Hot stoves, scalding liquids, worn-out fuses and electric cords, cigarettes and fires, open pools, falling out of car, backing car over child, and unlocked cupboards.

When a child is one year of age he begins to get into things, and by the time he is two years old there is nothing in the house that he cannot reach. Put things out of reach? There is no "out of reach" for the aggressive child of two years. If some children are to be permitted to poison themselves, why select only the alert and aggressive ones? A locked cupboard in so convenient a place that it can be used for all dangerous drugs makes a splendid gift for a child's first birthday. (This advice for a locked cupboard has been so effective that, in the author's practice, the need for stomach pumping has decreased from a former incidence of ten to twenty a year, to one in two years.)

All the counselling of parents outlined here takes some little time but it also saves physicians a great deal of time where they can most appreciate it—in long, irrelevant telephone calls. The greatest dividend of all is the feeling of satisfaction from rendering a good and useful service and the appreciation of satisfied parents.

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Paravaginal Hematomas

Their Recognition and Management Postpartum

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• *Paravaginal hematoma is an unavoidable obstetrical complication, usually due to the traction and rupture of paravaginal veins by the oncoming presenting part. Pain, swelling, ecchymosis and urinary retention are the usual symptoms if the hematoma is located below the levator plate, while supralevator hematomas give no external manifestations.*

Early diagnosis could be made more often if digital examination of the pelvis were done routinely after the third stage of labor and before dismissal of the patient from the hospital, and done more frequently in postpartum patients with pelvic complaints.

Active surgical intervention is advocated to avert needless destruction of tissue, prolonged morbidity, and delayed recovery.

HEMORRHAGE into the paravaginal soft tissue with subsequent hematoma formation is a serious and not uncommon obstetrical complication. It is the most frequent cause of covert postpartum hemorrhage and a formidable contributor to puerperal morbidity with delayed convalescence. Subperitoneal hematomas not associated with lesions of the uterus (so well considered by Williams[2]), and broad ligament hematomas from rupture of the uterus will not be included in this report, which is an inquiry into paravaginal hematomas which appear to develop from the passage of the oncoming presenting part through the vaginal canal.

ANATOMY OF THE PARAVAGINAL SPACES

The paravaginal space is that division of the pelvic connective tissue which extends from the vagina to the obturator fascia. Since it is contiguous with the paravesical, parametrial and pararectal spaces, blood (or exudate) arising in one connective tissue space may readily spread to any other. (The most complete anatomic and pathologic descriptions of these spaces may be found in the monumental work of Freund.[1])

A vaginal venous plexus surrounds the vagina, along the lateral border of which it is most prominent, because of the condensation of the vesico- and rectovaginal fasciae. One or more vaginal veins issue from the upper end of the vagina bilaterally, terminating in the hypogastric veins. The vaginal venous plexus intercommunicates with the inferior and middle hemorrhoidal veins as well as with the inferior vesical plexus, and this entire venous pool becomes tremendously engorged during the latter months of pregnancy.

The levatores ani muscles divide the paravaginal space into an upper or supralevator fossa and a lower or infralevator fossa. A paravaginal hematoma is typically confined to the upper or lower compartment, although massive hemorrhage can break through the levator barrier. Continued bleeding into one of these closed spaces causes "glacier-like movement" of the integument upon the subjacent tissue.

Hemorrhage into an infralevator space is characterized by massive swelling and ecchymosis of the labia, perineum and lower vagina on the affected side (Figure 1), and suggillation may extend over the buttock. Anorectal tenesmus may result from extension into the ischiorectal fossa, while urinary retention may succeed spread ventrally into the paravesical space. Supralevator hematoma, on the contrary, is not visible externally, and can only be diagnosed by digital examination of the pelvis. It can be felt as an insensitive rubbery mass pushing in the vaginal wall and more or less occluding the canal (Figure 2).

CLINICAL MATERIAL

In a series of 930 consecutive vaginal deliveries observed by the author, there were seven paravaginal hematomas, an incidence of one in 135 parturitions. The youngest patient was 20 years old, the oldest 39, and the average age was 32. Five women were primiparas and two were secundiparas. All pregnancies were uneventful, labor was not unusual, and all deliveries were by low forceps and episiotomy. Anesthesia was by spinal infusion in four cases, by continuous caudal in two, and by gas-

Presented before a Joint Meeting of the Sections on General Practice, Pediatrics and Obstetrics and Gynecology at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

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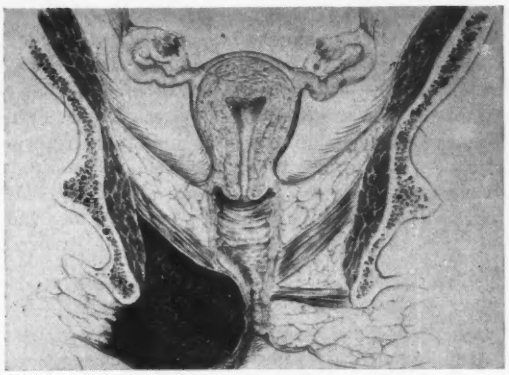

Figure 1.—Frontal section of pelvis to show hematoma in infralevator space. Note massive swelling of vulva, perineum and distal vagina on affected side.

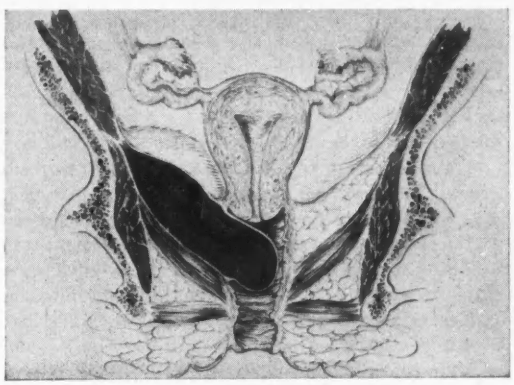

Figure 2.—Frontal section of pelvis to show hematoma in supralevator space. Note the medial displacement of the lateral vaginal wall almost to the opposite side, and the upward "tenting" of the pelvic peritoneum.

oxygen-ether in one. The infants were normal. The largest weighed 8 pounds (3,628 gm.).

Of the seven hematomas, four (including one in the perineum) were infralevator and three were supralevator in location. There was no maternal mortality.

EPISIOTOMY SITE AND LOCATION OF HEMATOMA

Table 1 summarizes the location of the hematomas as far as the site of episiotomy is concerned. It will be noted that in only one of the patients (Case 2) did the hematoma develop in proximity to the episiotomy wound. In the six other patients the location of the hematoma was quite distant from the site of episiotomy. It may be concluded that, in most instances at least, the formation of a paravaginal hematoma is not a complication of episiotomy.

CLINICAL MANIFESTATIONS

The salient clinical manifestations observed are summarized in Table 2. It will be noted that a mass

TABLE 1.—*Episiotomy site and location of hematoma*

Case	Episiotomy	Hematoma
1	Left medio-lat.	Right infralevator space
2	Median	Perineum
3	Median	Right infralevator space
4	Median	Right supralevator space
5	Median	Left supralevator space
6	Right medio-lat.	Left infralevator space
7	Median	Right supralevator space

TABLE 2.—*Clinical manifestations in seven cases of paravaginal hematoma*

Manifestation	No. of Patients and Position of Lesion		
	Supra-levator	Infra-levator	Total
Mass or swelling	3	4	7
Pain	..	3	3
Suggillation	..	3	3
Shock	1	1	2
Fever, septic	..	3	3
Urinary retention	1	3	4

or swelling developed in every case, and agonizing anogenital pain with suggillation of the vulva, perineum and buttock occurred in three patients. Two women went into moderately profound shock as soon as evacuation of the hematoma was undertaken, and emergency transfusion was necessary in both instances. Three women with sublevator hematomas had high fever for several days, that is, until surgical drainage was carried out. Stubborn urinary retention was an outstanding symptom in four patients, including one with a supralevator hematoma, until evacuation was achieved.

TIME OF DIAGNOSIS

Paravaginal hematoma was diagnosed in three of the seven cases in the delivery room and appropriate treatment was started without delay. Secondary bleeding in an infected median episiotomy wound rather suddenly caused a large hematoma on the fourth postpartum day in one patient. Despite continued pain, difficulty with micturition and unexplained fever, the diagnosis of infralevator hematoma was not made in two patients until ecchymosis of the buttock developed on the eleventh postpartum day in one case and the sixteenth in the other. In another case a huge supralevator hematoma which practically closed the upper vagina was not discovered until routine examination 42 days postpartum, and the patient was quite asymptomatic, her only postpartum difficulty having been urinary retention with residual urine for the first five days.

Earlier diagnosis should result from a wider knowledge of the frequency and manifestations of paravaginal hematomas following delivery. It is recommended that digital examination of the pelvis (rectally or vaginally) be done (1) before the patient leaves the delivery room, (2) whenever unexplained anogenital pain, ecchymosis, fever or uri-

TABLE 3.—*Summary of treatment in seven cases of paravaginal hematoma*

	Cases
Conservative treatment (misdiagnosis)	1
Surgical treatment	6
Evacuation and pack	3
Evacuation and suture	1
Evacuation, suture and pack	1
Evacuation, suture and secondary perineorrhaphy	1

nary retention develops postpartum, and (3) before dismissal from hospital.

TREATMENT

Early diagnosis and active therapy are necessary if needless destruction of tissue, prolonged morbidity and delayed convalescence are to be avoided. The obstetrical attendant must be alert to the fact that paravaginal hematoma may occur after the simplest delivery, and it usually has no physical or causal connection with the episiotomy wound.

The summary of treatment used is presented in Table 3. Active treatment (which always included surgical evacuation) was carried out in six patients, but withheld in one instance because a supralevator hematoma was mistaken for a Gartner's duct cyst. This undrained lesion took four months for resorption, during which time dyspareunia persisted. Transfusions were used to combat shock and to correct puerperal anemia secondary to blood loss and infection. All patients surgically treated received antibiotics postoperatively.

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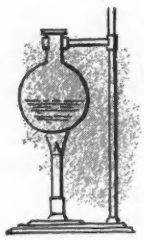

Air Pollution and Its Effect on Health

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• An experimental study of the effect of air pollutants on health can be undertaken only subsequent to the creation of synthetically polluted atmosphere in exposure chambers as a readily available and easily controllable laboratory tool. The many and varied pollutants must be studied singly and in combination so as to reproduce any synergistic or antagonistic effects that may exist. A study of pollutant substances at their source is wholly inadequate in view of the pronounced photo-chemical activity in the atmosphere. The products of this activity may well be the significant ones insofar as morbid effects are concerned.

In the acute and subacute biological studies, both in vitro and in vivo systems are being used with the experimental progression being from the simple to the complex.

A PRESENTATION of the effects of air pollutants on health may be compared with a discussion of the effects of microorganisms on health; the agents concerned with both are apparently limitless. The many and varied atmospheric pollutants, their ubiquity, their ever-changing status in a physically and chemically dynamic atmosphere, and the variations in human response unite to make a study of their biological effects extremely difficult. Clinically, this is so even during transient disaster periods when pollutant concentrations are high, the emission sources are limited, and epidemiological surveys can be undertaken. Studies initiated at a time when epidemiologic efforts have proven valueless and when pollutant levels are below the threshold for the production of immediate clinical symptoms would appear to be well-nigh impossible of success. Experimental investigations appear equally difficult. A fundamental requirement for a research program of this type is the exact duplication of atmospheric pollutants in a readily available and easily controllable laboratory tool form. Only within the recent past have studies in this direction been successful. On both the clinical and the experimental levels, physiologic and pathologic studies fall far short of satisfaction because of the nonspecific and quite subtle changes that occur in response to pollutant exposure.

Historically, clamor and concern over air pollution have followed episodes in which geographic, meteorologic and industrial factors have united to produce transient intervals of high pollutant concentration characterized by severe symptoms affecting a significant percentage of the exposed population. Deaths have occurred in a smaller but still significant number of exposed residents.

Six acute episodes have now been recorded, two in the Meuse Valley, Belgium, in 1915 and 1930, respectively; two in Donora in the years 1945 and 1948; one in Poza Rica, Mexico, in 1950; and, the most recent, the great London fog of early December 1952. Sufficient data are available to make at least a partial critical study of four of these episodes: the 1930 Meuse Valley incident,[15] the second Donora episode of 1948,[1, 18] the Poza Rica investigation of 1950[17] and the London fog of 1952.[6, 7] It is of considerable interest, although somewhat disheartening, that the agents responsible for the morbidity and mortality were positively identified in only one of the four—the Poza Rica incident, which was the most limited and restricted of all. In Poza Rica the pollutant was hydrogen sulphide, an asphyxiating gas; the source was a recycling and sulphur-recovery plant; and concentrations of the pollutant were well above the maximum allowable concentration (MAC) of 20 parts per million (ppm). In none of the other incidents could a single pollutant be inculpated as either the only or even a critical toxic agent.

High on the list of suspected agents in the Donora and London episodes were sulphur-bearing compounds expressed as measured sulphur dioxide. Since the MAC of sulphur dioxide is 10 ppm, it is of interest to observe that the concentration of sulphur dioxide in Donora measured at the time the fog was lifting was .5 ppm,[22] and in London on December 7 and 8, 1952, the concentrations measured 1.399 and 1.339 ppm, respectively.[7] In the Meuse Valley episode hydrogen fluoride[16] was suspected as the principal pollutant; concentrations of 0.5 ppm were calculated (not measured) whereas the accepted MAC is 3 ppm. Sulphur dioxide, a subsequent entry into the field of suspicion in the Meuse Valley episode, was estimated to be present up to 8 ppm

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This investigation is being supported by a grant from the Field Investigations and Demonstrations Branch, National Cancer Institute, National Institutes of Health of the Public Health Service, and the Environmental Health Section of the National Institutes of Health of the Public Health Service.

Presented before the Section on Public Health at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

and concentrations of up to 40 ppm were considered as possibilities. There are, of course, no data available to substantiate these latter estimates. It should be quite apparent that since episodes characterized by time limits, restricted geographic locations, specific meteorologic circumstances, and abnormally high pollution concentrations have proven to be beyond exact analytical pinpointing both as to the toxic agents and their morbid effect, similar studies on subtoxicological levels of pollutants must be approached with great caution.

CLASSIFICATION

Pollutants created by man have united with natural geographic and meteorological conditions to produce a polluted atmospheric environment in Southern California of sufficient frequency, duration, and intensity that certain measurable effects on health may properly be anticipated. These effects are in all probability the result of the summation of pollutant activity rather than the direct response to a single pollutant substance. In planning a study of these effects the following questions must be considered. One, what is the physical state of the pollutant material: gaseous, gaseous-particulate (aerosol), solid-particulate (soot), or combinations of these? Two, what is the chemical nature of the pollutants at the time of emission and what new compounds form subsequent to the photochemical activity of the atmosphere? Three, is the health effect related to the physical and chemical nature of the pollutant or do allied factors enter, including, as studied by others, possible increased host susceptibility to upper and lower respiratory tract disease?[2, 24] Four, is the host response specific in terms of specific pollutants? Five, do various pollutants act synergistically or in combination to produce a morbid response since even in disaster the MAC of the toxic substances measured has with one exception never been reached? Six, is the effect solely one of addition of toxic substances to the atmosphere or is there a secondary factor such as interference with solarization, an item related to skin tumor morbidity and antirachitic activity?[3] Finally, is there a psychic effect, and if so, how can it be measured?

The host response to air pollution may arbitrarily be divided into three clinical types: the acute, subacute and chronic. During periods of abnormally high pollutant concentration, immediate clinical effects may be noted, ranging from eye and upper respiratory tract irritation through respiratory embarrassment with dyspnea and chest pain to extreme morbidity with ultimate death. This *entire* spectrum of symptomatology is usually manifest in most of the exposed population group, with the severity of symptoms tending to increase in proportion to the prior cardiorespiratory disability of the exposed persons. The mass effects are transitory and disappear with a decrease in pollutant concentration to tolerated levels. It is of significance that in the Donora and London episodes the most severe illnesses and the greatest number of fatalities occurred in the older age groups and primarily among persons with heart disease, bronchitis, emphysema, bronchial asthma and pulmonary fibrosis. The milder symptoms, usually confined to exposed mucous membrane surfaces of the eyes and upper and lower respiratory tract, are as a rule limited to the healthy young and adult population groups. Retrospective studies following disaster periods indicate that a great many pollutant substances, active in an as yet undetermined cooperative manner, unite to produce the morbid effects described. Studies now going on indicate that specific pollutant host effects are transitory, especially in persons free of preexisting disease.

Subacute effects may be arbitrarily divided on the basis of the individual host under study. In the disease-free members of the exposed population, the subacute effects are characterized by sensory and cardiorespiratory symptoms that are more inconvenient than they are disabling. Lacrimation, rhinorrhea, cough, and occasional headache are all on the minimal clinical level and disappear with the disappearance of the abnormal pollutant concentration. The second type of response is that seen in exposed persons with antecedent cardiorespiratory disease. It would be extremely hazardous not to ascribe some progressive deleterious effect on an already impaired cardiorespiratory system by pollutants present most of the time. Even greater potential danger may threaten persons with marginally or critically decompensated cardiorespiratory systems, for they are conceivably capable of responding to very low concentrations. It is surely this group which is the primary source of deaths during extremely high concentration periods.

Cases of chronic or extremely delayed effect are even more of an arbitrary group than the former two. In effect, all people, without exception, may be responding on this level to the ever-present albeit ever-changing concentrations of atmospheric pollutants. In urban centers the exposure is for the entire life span, and it is this latter observation that must be considered in an assessment of the role of air pollution as one possible etiologic agent responsible for the increasing frequency of lung cancer. Certain epidemiologic observations direct suspicion toward such a relationship: first, the successful demonstration of known cancer-producing substances in the atmosphere and vehicular sources of pollution;[13, 14, 23] second, the experimental production of skin cancer in mice with air-extracted pollutants;[13] third, the reported greater incidence of pulmonary cancer in urban than in rural residents;[12, 21] fourth, the dif-

ferent rates of acceleration of incidence in various localities;[10] fifth, the variations in incidence in the two sexes from country to country;[11] and sixth, and perhaps most significant, is the presence of substances which, although in themselves of questionable carcinogenicity, are considered as providing a mechanism for the biological activity of the known and suspected carcinogens in the atmosphere.[13]

EXPERIMENTAL

The Los Angeles atmosphere, polluted primarily by a hydrocarbon, is characterized chiefly by its pronounced oxidizing capacity.[8] At present the accepted method of measuring pollutant concentration utilizes this chemical observation. Both chemical and biological systems are being used. The former measures total oxidants in the air, and the latter depends on the demonstration of specific morphological changes in certain susceptible botanical species.[4, 9] It seemed proper that in an experimental program to determine the biological effects of atmospheric pollutants in Los Angeles County the oxidation effect should be the first to be studied, particularly in light of the fact that eye irritants have been reported to act on chemical groups common to a number of significant enzymes and enzyme systems. The sulfhydryl group has been demonstrated as the site of the oxidant effect.[5]

The investigational approaches undertaken included (1) protein and enzyme studies to assess pollutant effects on plasma proteins and on enzyme and enzyme systems; (2) respiratory physiology studies utilizing pulmonary function measurements and blood gas analyses; (3) amino acid studies to indicate protein structure changes through the knowledge of the fate of amino acids; (4) hemoglobin studies to determine pollutant effects on oxyhemoglobin and nitric oxide hemoglobin, methemoglobin and sulfhemoglobin formation; (5) pathologic studies to demonstrate morphologic changes in the respiratory tract and other organ systems following pollution exposure and; (6) electrolyte studies to measure changes in blood concentration and electrolyte excretion.

Artificial smog used in this study was prepared according to the method of Shepherd[19] of the National Bureau of Standards and Haagen-Smit of the California Institute of Technology. In the course of analyzing the artificial smog, it became evident that it varied in a manner similar to that of natural smog which changes with the time of day, the geographic location of sample collection, and varying meteorologic conditions. It soon became apparent that the first step would be to carry out a series of repeated determinations so as to observe the entire life cycle of smog from the pollutant source through its build-up and finally to its destruction.

An atmosphere with 4 ppm total oxidant was arbitrarily chosen as the initial exposure concentration. This amount represented ten times the total oxidant, 0.4 ppm, measured in downtown Los Angeles at noon on a smoggy day. Unless otherwise stated, the 4 ppm concentration was used for all studies in this discussion. In instances where neither in vivo nor in vitro changes could be demonstrated at the 4 ppm level, the naturally occurring oxidant level of 0.4 ppm was increased approximately ten thousandfold to 3,000 ppm of oxidant in the atmosphere used for testing. Throughout the entire study it was repeatedly noted that additional tests (which are now being done) would be necessary to further establish the equivalence of natural and synthetic smog.

The biologic studies were planned on the basis of increasing complexity of test systems and all the studies were in the chemical, biochemical, physiologic and pathologic disciplines. The division is one of convenience only, as all studies overlapped. Chemical studies were initially performed for the double purpose of first, determination of smog-sensitive compounds and, second, establishment of the need for air-conditioned laboratories so that the natural atmosphere would introduce no artifact into the data.

Twenty-one amino acids were exposed to 4 ppm of oxidant for 17 hours to measure the changes that occurred. Eight of them—histidine, methionine, tyrosine, tryptophane, lysine, leucune, proline, cystein and glutathione—were found altered. The reaction products were separated from the parent amino acids by filter paper chromatography and detected by the ninhydrin color reaction and, when indicated, by absorption spectroscopy. These studies are as yet incomplete, and the reaction products have not been identified. These experiments are also being undertaken quantitatively.

Investigation of several of the vitamins produced variable results. Vitamin C was not tested, as its instability is well known. Thiamin and pyridoxine were altered by exposure to 4 ppm of smog oxidants for three minutes. Folic acid, carotene and alpha-tocopherol were altered only when exposed to the 3,000 ppm oxidant environment, at which concentration riboflavin and niacin were still unaltered. Cholesterol was oxidized following exposure to the 4 ppm smog for three minutes.

Among the nonprotein hormones it was found that the androgenic, progestational and adrenocortical hormones were destroyed following three minutes of exposure to smog with 4 ppm oxidant. The estrogenic hormones alone withstood all concentrations of smog.

Xanthine and uracil in the purine and pyrimidine group were tested, and alteration could be demonstrated only by exposure to high concentration of

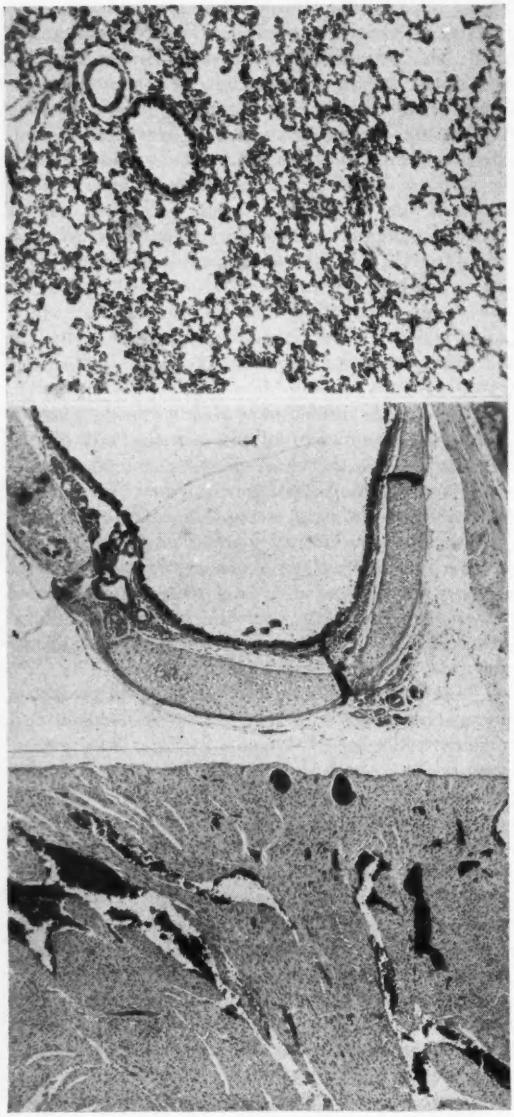

Figure 1.—*Upper:* Lung essentially normal. *Middle:* Trachea shows mild inflammatory response. *Lower:* Myocardium shows intense vascular congestion with focal areas of hemorrhage. (Magnifications × 30.)

3,000 ppm oxidant. In the porphyrin group, cytochrome-C and hemoglobin were significantly altered —30 per cent destruction of the former and 40 per cent methemoglobin formation in the latter. These observations were immediately checked in an in vivo system. Wistar white rats were exposed to smog of 4 ppm oxidant concentration for from 10 to 180 minutes. Hemoglobin oxidation to methemoglobin varied from 0.5 per cent to 10 per cent. In this group of experiments oxides of nitrogen were excluded from the smog mixture so that the oxidant effect could be determined independent of the known nitrogen oxides effect on hemoglobin. Increasing the smog concentration to 3,000 ppm oxidant killed the rats in less than ten minutes. Under these conditions up to 25 per cent methemoglobin was found and the plasma proteins gave altered filter paper electrophoretic patterns with an increase in the gamma globulin fraction.

Enzymatic studies have thus far been limited to the observations of succinic dehydrogenase activity in the liver of normal rats in a medium containing smog oxidants, and in livers of intact animals killed by smog. Data from these studies are as yet incomplete.

Physiological studies on cardiorespiratory activity, using large animal species with and without induced pulmonary disability, have not extended beyond the stage of control determinations of blood gases and the residual content of expired air.

Pathological changes demonstrable by standard histopathological methods proved to be nonspecific following exposure of rats and mice to tenfold the concentration of natural smog. Following a build-up of lethal concentrations to 3,000 ppm oxidant, morphologic findings of respiratory irritation associated with changes of nonspecific asphyxia were noted. Lacrimation and rhinorrhea developed in rats and mice during the build-up of lethal concentrations, as anticipated by the findings of Haagen-Smit. Pollutants were studied singly as well as in atmospheric-occurring combinations. Figure 1 shows the lung, trachea and myocardium of a rat killed by gasoline vapors in an inhalation chamber. Only the myocardium showed changes—pronounced vascular congestion characteristic of asphyxia alone.

The effects of nitrogen oxides on the lung—pronounced vascular congestion, alveolar congestion, hemorrhage and protein exudation into the bronchi, are shown in Figure 2. In Figure 3 are shown changes following exposure to smog with a concentration of 3,000 ppm oxidant. The trachea shows marked inflammation, edema, cellular infiltration and epithelial separation. The lung shows the most intense alveolar and vascular congestion with numerous focal hemorrhages.

To date, acute morphological changes have been significant only in those animals exposed to ten thousand times the concentration of pollutants that exists in Los Angeles atmosphere. Biochemical and chemical changes, however, have been demonstrated following short-term exposure to only tenfold the Los Angeles smog concentration. As yet, a critical appraisal of the acute and subacute data is impossible, as so many of the systems studied have been studied under circumstances that, to say the least, are unphysiologic. Further studies are indicated for an understanding of the mechanisms whereby the

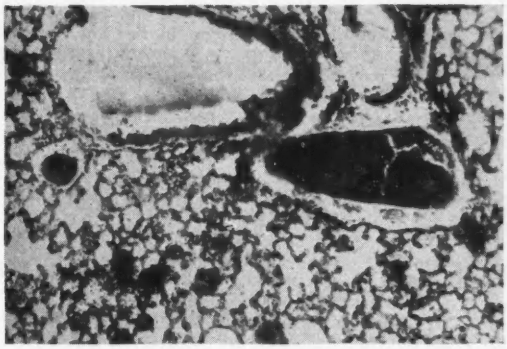

Figure 2.—Lung shows intense vascular congestion. The alveolar septa are engorged, and focal areas of alveolar hemorrhage can be noted. (Magnification × 65.)

TABLE 1.—*Atmospheric samples collected in Los Angeles*

Compound	Amount in mg. Total	Amount in mg. Per 1,000,000 Cubic Feet
Sample 1:		
Pyrene	0.28	0.14
3, 4-benzpyrene	1.84	0.92
1, 12-benzperylene	1.45	1.00
Sample 2:		
Pyrene	0.9	0.32
3, 4-benzpyrene	2.35	0.84
1, 12-benzperylene	0.72	0.35

Sample 1: August 1 to October 15, 1952—42 days actual sampling.

Sample 2: October 21, 1952, to June 1, 1953—59 days actual sampling.

many observed clinical effects are brought about. These include eye irritation, upper respiratory tract and lower respiratory tract irritation, and interference with normal respiratory functions in persons with decreased cardiorespiratory reserve.

The experimental approach to the investigation of the chronic or carcinogenic effect of atmospheric pollutants presents all the difficulties mentioned thus far, plus two critical additional ones. First, there is the necessity for maintaining the animal species for a long time; second, extrapolation of data on carcinogenesis in animals to imply kindred effect in humans is unwarranted.

Initial studies consisted of air sampling for the collection of material for chemical analysis and biological use. In the first series of experiments the collected material was used for skin painting on C57 black mice.

The air sampling revealed the presence of known carcinogenic hydrocarbons in the Los Angeles atmosphere (Table 1).

In addition to the aromatic polycyclic hydrocarbons demonstrated in the atmosphere, the presence of aliphatic hydrocarbons and their oxidation products is offered as being of significance in the pathogenesis of lung cancer in addition to their role as

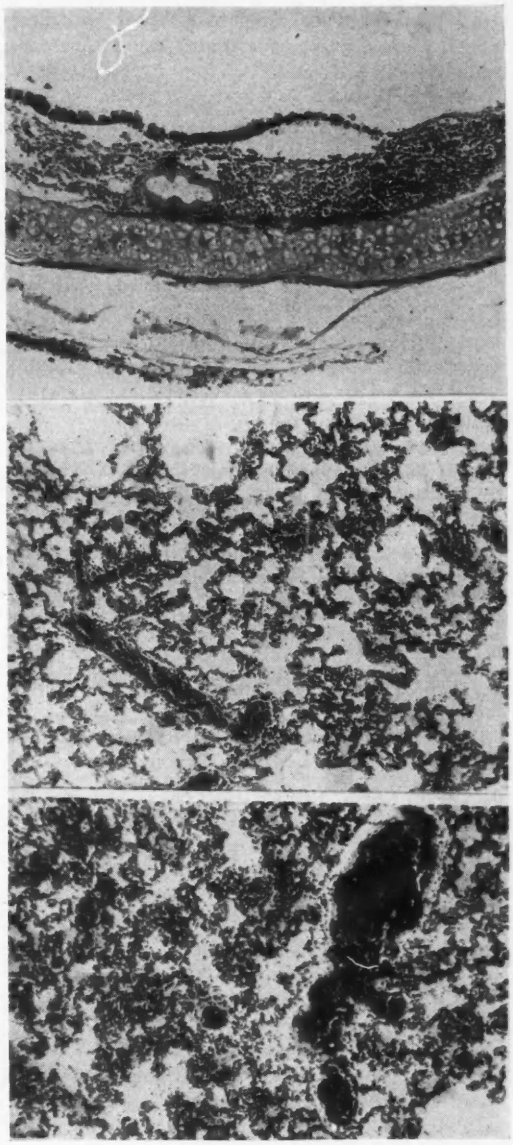

Figure 3.—*Upper:* Subepithelial inflammatory cell infiltration with edema with epithelial separation from underlying cartilage. *Middle:* Diffuse pulmonary congestion with alveolar hemorrhage and inflammatory exudation. *Lower:* Another section from lung showing pulmonary edema. (Magnifications × 65.)

nonspecific irritants. These highly polar substances are important, first, by providing an eluent for the separation of adsorbed carcinogenic hydrocarbon from soot particles in the air and, second, by the formation of various chemical compounds from unsaturated hydrocarbons, including (theoretically) the formation of diepoxides, which have carcino-

genic properties, according to a report on experimental work. Carcinogenic studies using these compounds are in progress.

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Pathology of Cat-Scratch Disease

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• A pathologic and histogenetic study of material obtained from ten cases of cat-scratch disease was carried out. The earliest lesion was of ten days' duration and the oldest of 35 days' duration. The first changes in lymph nodes consisted of proliferation of epithelioid cells followed by exudation of leukocytes in their centers and subsequent necrosis of the exudate and epithelioid cells. Proliferative changes leading to formation of epithelioid cell tubercles were seen in some cases. Homogenization of necrotic centers brought about the formation of caseous tubercles similar to those seen in tuberculosis, syphilis, lymphogranuloma venereum and tularemia. Differentiation and the final diagnosis of cat-scratch disease rests upon correlation of histopathologic observations, clinical studies and specific skin tests.

LEE FOSHAY[12] in 1932 was first to recognize a disease entity characterized by a primary skin lesion and regional lymph node enlargement following the scratch of a cat. His observations were not published. In a communication in 1952 he said that in 1945 Rose prepared an antigen from a diseased lymph node of a person who had this disease.[13] Later this antigen was used by Foshay as testing material on his own case. In 1950 Debre[8] published a report on this disease in France. Flores[11] cited the work of Petzetakis of Greece who in 1935 reported on the clinical and anatomical characteristics of this disease; and since that work antedated the publication of Debre, Flores called the entity *Petzetakis disease.* The first report in this country was that of Greer,[16] who in 1951 described a case in a young man. Positive skin reactions were obtained with an antigen supplied by Foshay. Since then many reports have appeared in this country (summarized in 1951, 1952 and 1954 by Daniels and MacMurray[5, 6, 7]) and in France, South Africa, Canada, Australia, England, Switzerland and Germany. In California several cases have been reported in scattered parts of the state (Cuttle,[3] Epstein,[10] Frank and Harder,[14] Gifford[15] and Todd[25]).

The clinical epidemiologic and etiologic features are well covered in the monographs of Daniels and MacMurray,[5, 6, 7] Mollaret and associates,[19, 20, 21] and Cuttle.[3] The disease is self limited, characterized by a primary skin lesion at the site of a scratch in most instances attributed to a cat, followed by swelling of regional lymph nodes after an interval of four days to a month. A fever of moderate degree may develop with malaise and loss of appetite. The nodes are tender and the skin may be reddened. The degree of enlargement varies, the nodes at times reaching 6 to 8 cm. in diameter. One or more regional nodes may be involved. Suppuration and sinus formation may occur although in milder forms the nodes may slowly involute without breaking down. The enlargement may persist for many months. A history of cat-scratch is obtained in the majority of cases. Dog bite, thorn scratch and rabbit bite have been reported as presumably the source of infection. In a small proportion of cases there is no history of skin injury. Complications are rare. Subcutaneous lesions suggestive of erythema nodosum have been described.[21, 24] Cases in which Parinaud's oculoglandular syndrome occurred also have been reported.[2, 9] Mild encephalitis was reported by Stevens.[24] Blood findings are not diagnostic.

The diagnosis of cat-scratch disease is based on the history, clinical course and laboratory tests. Exclusion tests for infectious mononucleosis, brucellosis, tularemia, tuberculosis and lymphogranuloma venereum should be done in all cases. A complement fixation test using lygranum as the antigen has been utilized by Mollaret and his associates[22] with positive reactions, indicating the close relation of this disease and lymphogranuloma venereum. By far the most important test available at present is a skin test using a 1:5 saline solution dilution of heat-treated exudate obtained from suppurative lymph nodes of known cases. This antigen, although crude, appears highly specific. Daeschner[4] emphasized the specificity of this antigen and the absence of reaction in nomal controls. This was confirmed by Bettley and Fairburn.[1] The skin reaction may remain positive for many years after recovery.

The cause of the disease is probably a virus, as yet not isolated, closely related to the psittacosis-lymphogranuloma venereum group. Mollaret and co-workers[22] were able to transmit the disease to monkeys but failed to grow the virus in tissue culture or transmit the disease to other animals. These investigators also described the presence of intra-

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Presented before the Section on Pathology and Bacteriology at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

TABLE 1.—*Clinical data on ten cases of cat-scratch disease*

Case	Sex and Age	History of Cat-Scratch	Primary Lesion	Site of Lymph Node Swelling	Duration of Swelling	Fever
1.	Male, 6 yrs.	Right hand	At site of scratch	Rt. axilla	35 days	None
2.	Female, 3½ yrs.	Numerous cat scratches	None	Rt. groin	30 days	Max. 102° F.
3.	Female, 53 yrs.	Numerous over arm	Right index finger	Rt. axilla	21 days	None
4.	Male, 60 yrs.	Repeated	Right hand	Rt. axilla	12 days	None
5.	Male, 35 yrs.	Numerous—both arms	Forefinger right hand	Lft. axilla	35 days	None
6.	Female, 42 yrs.	Many scratches	Left thumb	Sup. cubital nodes, lf.	10 days	Mild
7.	Female, 30 yrs.	Frequent cat scratches	None	Both inguinal nodes	27 days	Mild
8.	Female, 4 yrs.	No history of scratch	Right mandible	Angle of jaw	30 days	Mild
9.	Female, 43 yrs.	Frequent cat scratches	None	Rt. groin	21 days	Mild
10.	Male, 8½ yrs.	Cat-scratch of face	Left temple	Angle of lft. jaw	25 days	Slight

TABLE 2.—*Laboratory data on ten cases of cat-scratch disease*

Case	Frei Test	Tuberculin Test	Culture Lymph Node	Skin Test—Cat-Scratch Antigen	Heterophil Antibody	Agglutination Test—Tularensis	Agglutination Test—Brucella abortus	Blood Wassermann
1.	Neg.	Neg.	Neg.	3+	Neg.	Neg.	Neg.	Neg.
2.	Neg.	Neg.	Neg.	4+	Neg.	Neg.	Neg.	Neg.
3.	Neg.	Neg.	Neg.	2+	Neg.	Neg.	Neg.	Neg.
4.	Neg.	Neg.	Neg.	2+	Neg.	Neg.	Neg.	Neg.
5.	Neg.	2+	Neg.	3+	Neg.	Neg.	Neg.	Neg.
6.	Neg.	2+	Neg.	2+	Neg.	Neg.	Neg.	Neg.
7.	Neg.	Neg.	Neg.	4+	Neg.	Neg.	Neg.	Neg.
8.	Neg.	Neg.	Neg.	3+				
9.	Neg.	Neg.	Neg.	2+	Neg.	Neg.	Neg.	Neg.
10.	Neg.	Neg.	Neg.	3+	Neg.	Neg.	Neg.	Neg.

cytoplasmic inclusions which are similar to the elementary bodies and granular corpuscles of psittacosis. Wegmann[26] and Winship[27] expressed doubt that these intracellular granules are virus inclusion bodies.

As to the source of the etiological agent, little is known. Cats appear to be immune to inoculation and are usually healthy at the time of transmission of the disease. This fact prompted Mollaret to postulate that the cat serves to carry the disease from some other reservoir in nature, presumably birds. Much remains to be done to clarify problems of etiology and epidemiology.

The basis of the present communication is pathologic studies of lymph nodes and subcutaneous tissue in ten cases observed at Sutter Hospital in a period of 18 months. Eight additional cases of probable cat-scratch disease were observed but were not included in this study because they did not meet all the diagnostic criteria. In Tables 1 and 2 the pertinent clinical and laboratory data concerning these cases are given.

CLINICAL STUDIES

Only cases in which there were typical clinical history and positive reactions to skin testing with cat-scratch antigen were included in the present study. As was pointed out by Daniels and MacMurray,[5] weak or negative reactions to the antigen have been observed in cases of cat-scratch fever.

Four of the ten cases occurred in children under ten years of age; the oldest patient in the series was 60 years of age. On close questioning all gave a history of intimate exposure to cats. Nine of the ten had been scratched by cats. In seven of the ten cases a primary skin lesion was present. The onset of regional lymph node enlargement followed the primary lesion in from one to three weeks. In two instances the inguinal nodes were involved, in five the axillary nodes, in two the cervical lymph nodes and in one the superficial cubital lymph node. All the patients had surgical removal of the affected lymph nodes. The time between the first sign of lymph node enlargement and operative removal varied from 12 to 35 days. Mild fever was observed in six cases. Malaise, headache and loss of appetite were noted in few cases. In the majority of cases constitutional symptoms were mild or absent. Reports of blood counts were not included since they were not of diagnostic value. Slight leukocytosis (up to 14,000 cells per cubic millimeter) was reported, with slight rise in the percentage of neutrophils. Eosinophilia was not observed.

In all cases skin tests using Frei antigen (Lederle) gave negative reactions (Table 2). Tuberculin skin tests gave positive reactions in two patients. Cultures of involved lymph nodes were negative for bacteria and fungi. Heterophil antibody tests and agglutination tests with Brucella abortus and Pasteurella tularensis were negative. Results of Wassermann tests were negative in all cases. All patients had positive skin reaction to antigens of cat-scratch fever. With the exception of the patients in Cases 9 and 10, who were tested with an antigen prepared from one of the patients in the present series, these patients were tested with antigen supplied by Drs. Daniels

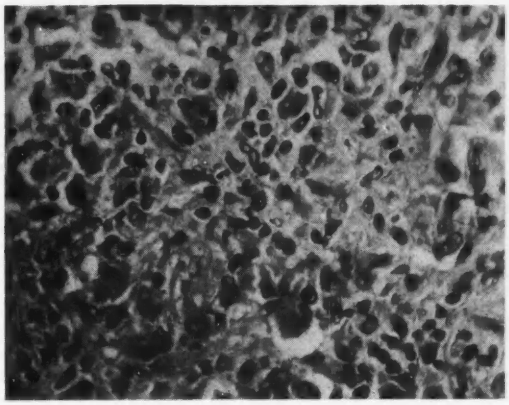

Figure 1.—Showing focal area of reticulum hyperplasia with beginning formation of proliferative tubercle (× 400).

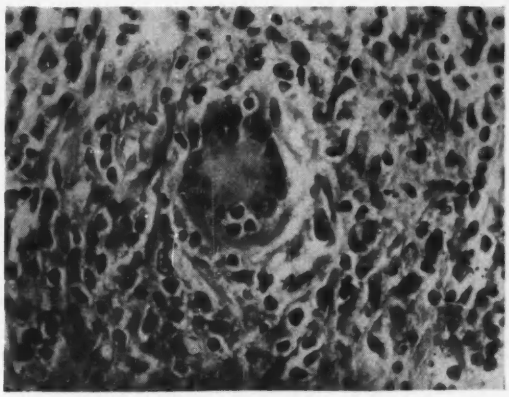

Figure 2.—Small tubercle with central Langhan's giant cell (× 400).

and MacMurray. Positive reactions were characterized at 48 hours by a slightly elevated indurated nodule of pale reddish-brown color, varying in size from 0.5 to 1.5 cm. with a larger zone of erythema about the papule.

All patients made uneventful recovery following surgical removal of the enlarged lymph nodes despite extensive suppuration of the nodes and subcutaneous tissues.

PATHOLOGICAL CHANGES

The lymph nodes that were removed were enlarged and firmly adherent to the surrounding structure. The overlying skin was reddened in the severe cases and the nodes were tender. A variable amount of thin creamy exudate was observed in six cases. In Case 7 about 30 cc. of exudate was aspirated from the lymph nodes and subcutaneous tissues. The lymph nodes with pronounced suppuration were removed piecemeal with fragments of surrounding tissues. In milder cases the lymph nodes were intact. The surfaces of the nodes were roughened and small satellite nodes were usually adherent. The cut surfaces of some of the specimens showed a background of pale gray tissue in which many small discrete pale yellow irregular foci were seen, ranging in size from 2 mm. to 1 cm. in diameter; others showed a moist cut surface of brownish red color in which were seen small opaque foci of gray to light yellow color. The lymph nodes removed after the disease was far advanced were considerably indurated.

Sections were stained with hemotoxylin-eosin, Mallory's methyl blue-eosin, Van Gieson's stain, periodic acid leuko-fuchsin reticulum method, trichrome stain of Masson, Mann's methyl blue-eosin, reticulum stain, Giemsa, Feulgen's stain, acid-fast stain, Gram stain and Macchiavello's stain. Sections and smears from all cases were examined for acid-fast organisms, pathogenic fungi and bacteria, with negative findings.

The earliest changes were seen in lymph nodes at ten days following the first signs of tumefaction (Case 6). These consisted of proliferation and swelling of reticuloendothelial cells to form irregular focal aggregates (Figure 1). The aggregates were most numerous in the cortex but the medulla was also involved. These cells, similar to the epithelioid cells seen in tuberculous lesions, had abundant eosinophilic cytoplasm, poorly defined cell borders and large pale-staining nuclei. Small clusters of these cells formed typical miliary tubercles composed of epithelioid cells, occasionally with a central giant cell of Langhan's type (Figure 2). In the centers of the medium-sized and larger aggregates of reticuloendothelial cells, exudation of polymorphonuclear leukocytes and fibrin occurred. In early lesions scattered lymphocytes appeared among the epithelioid cells; but in specimens taken at a later stage of the disease the epithelioid cells in the center of the lesion were replaced by dense masses of leukocytes, at first well preserved in structure but later showing degenerative changes of varying degree mixed with necrotic fragments of epithelioid cells (Figure 3). These degeneration changes were more pronounced at twelve days (Case 4). As a result, abscesses were formed with centers of necrotic cellular and nuclear granular debris surrounded by a well defined zone of epithelioid cells which tended to form a pallisade arrangement. Between the epithelioid cell layer and the central zone of necrosis an intermediate layer was usually present, consisting of semi-necrotic epithelioid cells. This layer was of variable thickness and composed of deeply eosinophilic granular debris containing attenuated pycnotic nuclei radiating toward the center. The shapes of the larger lesions were irregular and angular, very similar to the stellate abscesses seen in lympho-

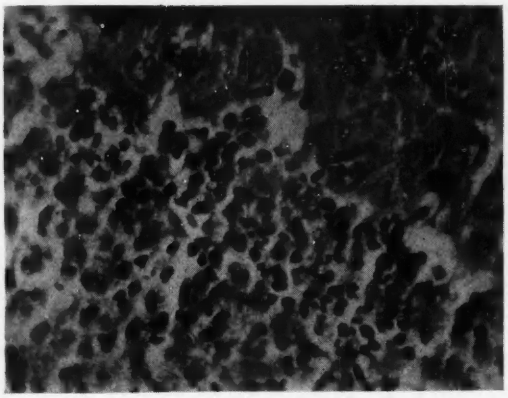

Figure 3.—Small micro-abscess with wall of epithelioid cells (× 400).

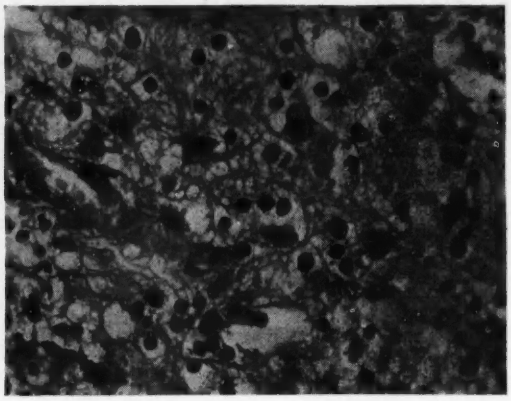

Figure 4.—Diffuse infiltration of histiocytes with foamy cytoplasm (× 400).

granuloma venereum. The smaller lesions were usually round in outline. In the center of the abscesses small remnants of epithelioid cells could be seen, and with the periodic acid reticulum stain the outlines of preexisting vessels were frequently noted. Reticulum fibers were present in the central zone of the early exudative phase but completely destroyed in the later stages.

The abscesses frequently showed conversion of the necrotic central mass into a homogeneous eosinophilic mass, at first patchy but later involving the entire central portion. Fairly well developed lesions of this type appeared at about 25 days. Complete homogenization of the centers of the abscesses was seen only in lesions of 30 to 35 days' duration. With Van Gieson's stain the homogeneous centers stained pale yellow; and when the homogenization was fully developed the staining reaction and appearance of the lesions were identical with the caseous centers of tuberculosis lesions. In well developed caseous lesions four zones were usually present, namely a central zone of caseous necrosis, a thin zone of partial necrosis, a multilayered zone of epithelioid cells arranged in pallisades and an outer layer of epithelioid elements arranged circumferentially. About these lesions there were large numbers of plasma cells, moderate numbers of eosinophils and many lymphocytes. Reticulum stains showed a delicate reticulum about the epithelioid cells and coarser fibers and collagenous fibers forming a network at the periphery. In the areas of early exudation considerable reticulum was present which disappeared as these foci became necrotic. However, a zone of collapsed reticulum was usually seen in the region of the junction of the necrotic center and the epithelioid cell layer. With the periodic acid reticulum stain a rather rich vascular network was demonstrated which extended through the zone of epithelioid cells toward the zone of necrosis.

Some of the epithelioid foci did not undergo central necrotic changes but persisted as irregular groups of cells which later showed formation of considerable amount of intracellular collagenous fibers. These changes were clearly demonstrated by Van Gieson's stain. In most cases small proliferative miliary tubercles were demonstrable.

An inflammatory reaction extended into the surrounding capsule and fatty tissue of varying degree in all cases studied. Mild involvement consisted of dense foci of lymphocytes, eosinophils and plasma cells, usually perivascular in arrangement. The small veins and arteries showed leukocytic infiltration of the outer layers, fraying of the collagenous fibrils and occasional small thrombi within their lumens. Frequently the tissues about the lesion showed extensive diffuse inflammation with formation of large abscesses usually surrounded by a thin layer of epithelioid cells. Diffuse sheets of histiocytic elements were frequently seen (Figure 4). These often showed abundant foamy vacuolated cytoplasm with sharply defined cell margins. Later fibroblastic proliferation occurred, accompanied by marked proliferation of capillaries. Plasma cells were very abundant. Numerous giant cells of foreign body type were seen in areas of diffuse inflammation. In addition, giant cells of Langhan's type were seen in the wall of the large abscesses. Some of these contained nuclear debris and were located on the inner epithelioid zone immediately adjacent to the central necrotic exudate.

In the older lesions (Cases 1, 5 and 8) extensive fibrous tissue proliferation was seen in the periphery of the necrotic foci surrounding the epithelioid cell zone and extending widely into the surrounding lymphoid tissue. In these areas of fibrosis, considerable infiltration of plasma cells were frequently noted. The lymphoid structures were largely replaced by fibrous tissue. The necrotic centers ap-

peared contracted and were surrounded by a thin layer of epithelioid cells and a wide zone of connective tissue. The radial arrangement of epithelioid cells was retained in some of the lesions and lost in others. Replacement of this layer with fibrous tissue was noted, but there was little evidence of organization of the central caseous areas in the material studied. Calcification was not observed in the necrotic foci.

Only the early stages of healing were present in the material studied (Cases 1, 2, 5 and 8).

Skin lesions. In one case the primary lesion of the skin involving the lateral surface of the right thumb was removed. This was a small indolent lesion of violaceous color with a small central crust. A small amount of thin exudate had been expressed from time to time. The duration of this lesion was one month.

Microscopic examination showed the presence of a widespread inflammation involving the corium and the adjacent subcutaneous tissue. Aggregates of lymphocytes, plasma cells and eosinophils were present about the sweat glands, hair follicles and the small blood vessels. Small miliary granulomas were seen directly beneath the epithelium and also in the deeper corium. These consisted of small foci of epithelioid cells among which were many lymphocytes and few eosinophils. One tubercle contained a central giant cell. The epithelial layer showed slight thickening and edema of the deeper layers.

Inclusion bodies. Whenever possible, smears were made from unfixed fresh tissues by scraping the surfaces with a knife blade and spreading the material over a glass slide. The smears were fixed in methyl alcohol, corrosive sublimate and by drying. These were stained by Giemsa, Macchiavello's and Feulgen's methods. In smears prepared in this manner a more satisfactory study of cellular inclusions was possible than with fixed tissue sections. With Giemsa stain intracytoplasmic bodies were seen, principally in large reticuloendothelial cells. These were in the form of irregular bodies, some rounded, others of crescent shape with a maximum diameter of about 3 to 5 microns. In addition to these larger bodies there were numerous small rounded forms of fairly regular size and about 0.2 to 0.3 microns in diameter. These stained a light pure blue in contrast to the violet blue of nuclear material. With Macchiavello's stain these bodies took both the blue and red stain; the periphery appeared blue and the central portion showed red coloration. In addition, minute bodies were scattered in the cytoplasm which stained bright red. Other reticuloendothelial cells showed the presence of minute discrete particles smaller than bacteria which were loosely scattered in the cytoplasm. These stained blue with Giemsa. In fixed material both the large and finely divided bodies were demonstrable in swollen reticuloendothelial cells. With Mann's stain the finely divided granules appeared light purplish red. These bodies did not take the Fuelgen's stain for desoxyribose nucleic acid.

DISCUSSION

The pathological changes in cat-scratch disease follow a fairly consistent pattern. In study of a number of cases including both early lesions and fairly advanced lesions, the histogenesis of the lesions became apparent. The earliest changes consisted of reticuloendothelial hyperplasia in the form of plaques and small focal clusters These foci further developed in one of two directions. The most common form was characterized by exudation of polymorphonuclear leukocytes within their centers with the formation of microabscesses, some rounded and others stellate. These abscesses were surrounded by a well-defined zone of epithelioid cells tending to pallisade. Less frequently the small aggregates of epithelioid cells did not undergo necrosis but developed into tubercles of proliferative type, many containing central giant cells. These were seen in both the skin and lymph node lesions and often coexisted with suppurative foci.

The abscesses varied considerably in size and often reached large dimensions, especially in the tissues surrounding the involved lymph node. Pronounced degenerative changes occurred in the exudate with the conversion of the exudate into a basophilic granular mass containing nuclear and cellular debris. This granular debris later became hyalinized and stained pink with hematoxylin-eosin and light yellow with Van Gieson's. In this manner solitary and conglomerate tubercles were produced which were often undistinguishable from caseous lesions of tuberculosis. These areas of caseous necrosis coexisted side by side with suppurative foci, and all stages of development were seen in multiple sections of lymph nodes and surrounding tissues in a single specimen. On the basis of cytology the differentiation between this lesion and tuberculosis was frequently difficult, and no doubt in the past many of these nodes were classified as tuberculous inflammations despite lack of demonstrable organisms. The most important finding which served to differentiate this lesion from tuberculosis was the coexistence of an extensive exudative process and proliferative lesions, the former predominating in most instances. However, there were examples in which tubercles, both miliary and solitary, predominated and in these cases differentiation on the basis of morphology alone was indeed difficult.

The histogenesis of the caseative lesions differs in some respects from that of tuberculosis. It is Huebschmann's hypothesis[17] that the earliest phase in the formation of the tubercle is an exudative re-

action associated with tissue injury. From a study of the earliest change in cat-scratch disease, reticulum hyperplasia is the earliest observed reaction; the exudative phase appears later and involves the center of the proliferative foci replacing these cells and forcing them toward the periphery. Necrosis and homogenization of the central exudate then follows. As in tuberculosis, caseous necrosis is bound to the exudative process. This process, however, does not follow the same pattern as Huebschmann describes in tuberculous lesions. It appears from these studies of the lesions of cat-scratch disease that the changes take the following order, namely, proliferative phase, exudative phase and finally the phase of coagulative necrosis, whereas in tuberculosis, according to Huebschmann,[17] the sequence is exudation-caseation-proliferation.

Mollaret and co-workers recognized three phases in the development of the lesions of cat-scratch disease. They observed that the first stage, or "elementary phase," consists of proliferation of reticuloendothelial cells, the second, or "accentuated phase," is characterized by exudation of leukocytes and formation of micro-abscesses and the third, or "ultimate phase," by formation of large foci of suppuration. Although this in general characterized the manner of development of the exudative process observed in the present series, this classification does not fully explain the development of the proliferative and caseative lesions so frequently observed. Also, all stages of development may be seen in the lymph nodes and surrounding tissues, indicating simultaneous regression and acute exacerbation. In addition, the process may stop at the first phase and heal by fibrosis.

It is difficult to reconcile the observations of the pathological changes in the material studied with those of Winship[27] who, in reporting upon a study of 29 cases, stated that epithelioid cells do not proliferate until abscesses are well developed, whereas the reverse appears to be the case. Also Winship's statement that changes simulating caseous lesions of tuberculosis were not present in cat-scratch disease cannot be accepted on the basis of observations made in material from the present study. In physical appearance and tinctorial characteristics, coagulative necrosis in cat-scratch disease is indistinguishable from the caseous necrosis of tuberculosis.

The differentiation of cat-scratch disease and lymphogranuloma venereum on the basis of gross and histological characteristics is extremely difficult. There is a greater tendency in lymphogranuloma venereum to suppurate and form multiple sinuses, although in cat-scratch disease this may be observed in severe cases. The distribution of the lesions is quite different. The nodes of the upper portion of the body, mainly the epitrochlear, axillary and cervical nodes, are most commonly involved with cat-scratch disease whereas the inguinal nodes are most frequently involved in lymphogranuloma venereum. Cytological changes are very similar; and the histogenesis is similar in the two diseases. The intermediate zone of partial homogenization of epithelioid elements appears much broader in lymphogranuloma venereum than in cat-scratch disease. In general the tendency to form caseative tubercles is greater in cat-scratch disease than in lymphogranuloma venereum. Despite these differences, the lesions cannot be separated on the basis of histological changes alone. The final diagnosis is dependent on the clinical studies, laboratory tests and skin tests. The striking similarity of the histopathological changes in the two diseases lends additional support to the theory that the causative organisms are closely related.

The ulceroglandular form of tularemia must be differentiated from cat-scratch disease. The early phases of tularemia show focal areas of necrosis bordered by narrow rows of epithelioid cells. Often the epithelioid reaction is insignificant or masked whereas in cat-scratch disease the epithelioid reaction is the initial reaction, with exudation and necrosis of their centers a later development. In the later stages the differentiation between the two conditions may be more difficult. Tubercles with caseous centers and epithelioid cells may develop in both diseases. Lillie and Frances[18] described an inner collagenous zone separating the caseous from the epithelioid pallisades. This is not commonly observed in cat-scratch disease. The clinical and laboratory studies further serve to differentiate these two conditions.

Other conditions such as sporotrichosis, tertiary syphilis, brucellosis and diseases caused by pathogenic fungi must be ruled out by appropriate studies.

The significance and diagnostic importance of intracytoplasmic inclusion bodies is debatable. With Macchiavello's stain these bodies are clearly demonstrable in two forms, namely large irregular plaques 7 to 10 microns in size, and small micrococcoid forms. A matrix was not demonstrable. Despite the fact that the tinctorial reaction and morphologic features of these inclusions are similar to those described in psittacosis by Yamamura and Meyers[28] and in cultures of virus of lymphogranuloma venereum by Rake,[23] final proof that these are specific inclusion bodies must await isolation of the virus and demonstration of similar inclusions in tissue culture.

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Anesthesia for Eye Operations in the Aged

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• *Aged persons appear to fear eye operations. Hence both patient and surgeon are more at ease with the use of a general anesthetic, particularly if the anesthetist has visited the patient before the operation and reassured him. Early ambulation, ordinarily so desirable in the aged, is curtailed in most eye operations because the head and eyes must be completely quiet. Since barbiturates are not well tolerated by aged patients, Dramamine is used instead preoperatively. Dramamine adequately sedates but does not cause depression or hallucinations. Morphine is contraindicated because of its pupillary action; Demerol is the drug of choice for preoperative medication.*

Xylocaine applied directly to the pyriform fossa and vocal cords prevents laryngospasm, coughing and straining. Then, following induction with Pentothal, a Guedel airway is introduced into the oropharynx and through it a continuous flow of oxygen is maintained throughout the procedure. Relaxation of the eyelids is aided by the use of various muscle relaxants, succinylcholine being the relaxant of choice because it is rapidly eliminated. By administering narcotics intravenously during the course of the operation the amount of Pentothal needed can be held to a minimum. To prevent any slowing of the respiration, Nalline is administered in conjunction with the narcotic. In dealing with debilitated patients, Nalline is usually given in conjunction with the preoperative narcotic. Any latent slowing of the respiration can be promptly relieved by an additional dose of Nalline.

MOST OPERATIONS ON THE EYES of aged persons are elective cataract extraction or iridectomy. Since the operations are elective, the usual problems of the aged, such as nutritional deficiencies and cardiovascular, renal and liver disorders have in most cases been medically surveyed and corrected as far as possible before the patient is admitted to the hospital.

Most elderly patients appear to fear operations on the eyes as much as they do major abdominal operations. This fear may account for the fact that there is a steady increase in general anesthesia for eye operations. Many surgeons prefer general to local anesthesia. With the patient asleep the surgeon has no urge to hurry as he might if the patient were awake and becoming restive and uncooperative. Moreover, the patient is relieved of the emotional strain of facing the operation and of fear that he might not be cooperative (many suspect that most failures are caused by poor cooperation).

Care of elderly patients after operations on the eyes presents special problems. Postoperative procedures ordinarily carried out after other kinds of operation—early ambulation, leg exercise, frequent turning in bed and encouragement of cough—have to be foregone. After cataract removal, for example, the patient must lie quietly flat in bed for from two days to a week, and for at least three weeks after repair of a detached retina. Coughing, straining, gagging or sneezing must be avoided. In patients of the older age groups the success of operation depends more on the quality of preoperative preparation, the administration of anesthesia and postoperative care than on the operation itself.

PREOPERATIVE INTERVIEW

Elderly patients are accustomed to certain habits and surroundings and the change of environment upon entering the hospital often gives rise to apprehension. Frequently an anesthetist deals with a depressed and discouraged patient in preoperative interview and an optimistic approach to the problem is welcomed. In the preoperative visit one should never be hurried and the visit should be one of pleasure and reassurance to the patient. Aged persons are usually eager to disclose previous anesthetic difficulties, allergic sensitivities and intolerance to certain drugs. Much can be gained from careful attention to their remarks. Many are particularly concerned about the kind of anesthetic they are to receive. Complete frankness and full explanation of their anesthetic problem is both desired and appreciated. They should be assured that there will be little discomfort or pain following the operation.

As a rule there is no need for preoperative sedation, but if it is needed codeine may be given. (To be certain the patient is not sensitive to codeine, a test dose of 30.0 mg. is given.) As Demerol® is administered both before and during operation, inquiry should be made regarding sensitivity to it. The pa-

Chairman's Address: Presented before the Section on Anesthesiology at the 83rd Annual Session of the California Medical Association, Los Angeles, May 9-13, 1954.

tient should be assured that although he will be awake on going to the surgery, preoperative medication will give a feeling of complete indifference and he will be calm and cooperative.

For sedation the night before operation, either Nembutal® or Seconal® is given in 50 to 100 mg. dosage. Since aged patients do not tolerate barbiturates well, they should not be given preoperatively the day of operation. Dramamine® instead is very effective; it does give rise to some drowsiness but does not cause hallucinations or respiratory depression and it reduces the incidence of nausea and vomiting.[11] With Dramamine the patient arrives in surgery awake, stolid and indifferent. Dramamine thereby meets one of the chief requirements of operations on the eyes in the aged—that of having the patient adequately sedated but not depressed. The dose of Dramamine is 50 to 100 mg. If administered orally it is given two hours before operation; if parenterally, it is given along with Demerol and atropine one hour before operation. Demerol, the narcotic of choice, is administered in 50 mg. dosage. Morphine is contraindicated for patients having cataracts removed because its pupillary action often counteracts the dilating effect of homatropine or Neo-Synephrine® and thus delays operation.

It is important to prevent straining, coughing, gagging and vomiting both during and after operation. The most effective means of accomplishing this is to anesthetize the sensory nerve ends of the larynx. This can be done in numerous ways and with a variety of anesthetic agents but the most consistent results have been obtained with topical application of 2 per cent Xylocaine solution. With the use of laryngeal forceps the solution is applied directly to the pyriform fossa and the vocal cords. The best indication that the anesthetic solution has been properly placed is difficulty in initiating the act of swallowing.

In order that the surgeon's access to the operative field may be unimpaired, it is customary to introduce the tube for intravenous drip at the wrist, the elbow or the ankle. Anesthetic solutions are then injected in the rubber tube portion of the intravenous set.

Pentothal sodium in 2 or 2.5 per cent solution is the agent used for induction of anesthesia. As soon as the patient is asleep the lower jaw relaxes, which, with the face of the patient straight up as it must be for operations on the eye, usually partially or completely obstructs the airway. A Guedel airway lubricated with 5 per cent Xylocaine ointment is therefore introduced into the oropharynx; with slight extension of the head, the airway then will be patent.

The multiple drapes used in eye operations completely cover the face with the exception of the operative field. To insure adequate oxygen and prevent accumulation of carbon dioxide beneath the drapes, a continuous flow of oxygen at a rate of 3 to 4 liters per minute is supplied by inserting a rubber catheter into the Guedel airway. This may stimulate laryngeal reflexes with resulting spasm, coughing or gagging if the topical anesthesia is inadequate.

Good relaxation of the eyelids and a quiet eye are required in eye operations. Some surgeons prefer blocking the seventh nerve, by either the Van Lint or the O'Brien method, for this purpose. Excellent relaxation of the lids and elimination of ocular movement can be obtained, however, by using one of the several muscle relaxants. The special value of the muscle relaxants lies in the selective sequence of muscle paralysis—the muscles of the eyes and lids first. Judicious use of a muscle relaxant not only eliminates the squeezing of the lids and movements of the eye but reduces intraocular tension. There is considerable variation among persons in sensitivity to the various muscle relaxants.[8] The only way to determine the effect of a given relaxant upon a patient is to observe the effects of the initial dose and the first supplementary dose. Succinylcholine is the drug of choice for this purpose in eye operations, for it acts quickly and any untoward effect is apparent within one or two minutes. Moreover, owing to rapid enzymatic hydrolysis of the drug, unwanted effect will abate within two to five minutes. Succinylcholine is administered by continuous drip at a speed that will maintain adequate lid relaxation with little respiratory depression.

In aged persons the amount of Pentothal® needed for induction and maintenance of anesthesia is almost impossible to predict. Even with small intermittent doses which are presumably metabolized rapidly there is such a wide variance in response that sometimes it takes a long time for even small doses to be eliminated. For the purpose of reducing the amount of Pentothal needed it has become a common practice to administer morphine or some comparable narcotic intravenously during the course of the operation. This intravenous opiate usually causes so much slowing of the respiratory rate that often it becomes necessary to assist respirations to insure adequate oxygenation. The slowing of respiration induced by morphine or similar opiate can be relieved by the administration of N-Allyl-normorphine (Nalline®).[2] When Nalline is administered before or in conjunction with the narcotic respiratory depression is prevented.[5, 6] Nalline probably displaces the opiate from certain receptors in the respiratory center in a competitive fashion.[6, 9] Although Nalline itself is slightly depressant to the respiratory center it is less so than morphine or similar opiates.[12] Displacement of the opiate by Nalline immediately eliminates the characteristic slowing effect of the opiate on the respiratory rate[2] and increases the sensitivity of the respiratory center to carbon dioxide.[3, 4] The increased concentration of carbon dioxide in the

blood stimulates the respiratory rate above normal for a few moments until the carbon dioxide is reduced toward a more physiologic level; then respiration becomes normal and remains normal.[5, 10] Nalline does not cause significant changes in blood pressure or pulse rate. It does not alter arterial oxygen content but does cause pronounced decrease in arterial carbon dioxide content.[10] The displacement action of Nalline is as effective with the derivatives of morphine — meperdine (Demerol), methadon (Dolophine®) and methorphian (Dromoran®)—as it is with morphine itself.[1, 7] It has no effect on the respiratory depression caused by Pentothal.[5, 6, 7]

Nalline is administered in conjunction with the opiate. It is prepared in the following manner: Nalline 10 mg. and Demerol 100 mg. (or morph sulphate, 15 mg.) are combined and diluted to a total of 10 cc. This mixture is administered intravenously in the amount desired. With patients aged 70 or older it is a common practice to administer 50 mg. of Demerol and 5 mg. of Nalline. Both Nalline and the opiate are usually eliminated in three to four hours but it must be kept in mind that in aged persons the narcotic elimination may be delayed, with resulting characteristic slowing of the respiratory rate. In a series of over 700 cases observed by the author there was one instance of delayed respiratory depression.

There is a considerable variation in response to narcotics in the aged, and sometimes when the Nalline-opiate mixture is administered to a person who already has received other drugs, slowing of the respiratory rate may occur. Additional Nalline will increase respirations. In this series there was no change made in the ratio of Nalline to opiate. Less Nalline might be as effective. With the one exception more Nalline was not needed.

The lethal dose of Nalline in man has not been clearly determined, but it is suggested that not more than 40 mg. should be administered in a single dose. The largest single dose administered in this series was 10 mg.

The author has observed 388 cases in which persons 70 years of age or older were given a Nalline-Demerol mixture for anesthesia during operations on the eyes. It was very satisfactory. Side effects were minimal. There was no increase in nausea or vomiting. Use of the mixture reduced the use of Pentothal, which decreased postoperative sleeping time.

It has long been believed that large therapeutic doses of narcotics are contraindicated for aged persons. It would appear that the concept of restricting the use of narcotics in the aged must be reevaluated in the light of the present experiences with the use of a Nalline-opiate mixture.

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Permanent Disability Evaluation

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• Physicians' reports for the purpose of permanent disability rating differ in character and in scope from usual clinical reports. Complete and precise reporting of permanent disability factors by physicians aids the Industrial Accident Commission in making proper awards for permanently disabled workmen.

The use of a CMA-approved method of reporting permanent disability factors reduces misunderstanding and needless delay in adjustment of cases.

THIS YEAR MARKS the fortieth anniversary of the California system of permanent disability evaluation of the Industrial Accident Commission and, generally, of the Workmen's Compensation Law as it stands today. This would therefore seem an appropriate time to review the basic principles of the California system, so that you the attending, treating and examining physicians of the state, and we of the Commission, might view the problem of permanent disability through the same eyes and thereby might better serve the people of California in our joint tasks.

First of all, it must be remembered that the concept of damages is alien to any consideration of workmen's compensation benefits. It was the inability of the damage system to cope with the problem of the industrially injured workman that led to the development of workmen's compensation. The purpose of the law is not to indemnify an injured employee for the consequences of injury, but merely to provide assistance to him during his period of disability. While it is a fundamental principle of workmen's compensation that industry should take care of its own, an injured employee must still bear some of the burden resulting from his injury.

Many methods have been developed for compensating permanent disability in the years that have elapsed since the first workmen's compensation principles were conceived. The founding fathers of workmen's compensation in California studied other existing methods and found them wanting.

One theory, that payment should continue indefinitely solely upon a percentage of actual wage loss following injury, was not accepted. Influences independent of the disability itself (such as, for instance, economic cycles, good times and bad) and individual factors (such as opportunity for obtaining employment because of friends or relatives, or, on the other hand an unwillingness to seek reemployment) quickly beclouded the effects of the disability itself. The necessity for making allowances for these extraneous factors and the necessity for following every case for years would have presented administrative problems of considerable magnitude.

Use of the so-called flat rate schedule composed of a relatively short list of disabilities and providing a specified amount of money or number of weeks of payments for each disability, left much to be desired. First, the injury list was meager and not detailed, leaving too many nonscheduled disabilities and with but few rules for evaluating them. The important elements of occupation and age were not included for the most part, or, if they were mentioned, no specific method of weighing these factors was indicated.

In 1914, the Commission brought forth a Schedule for Rating Permanent Disabilities. It was unique in character and concept. It demonstrated that the factors of disability, occupation and age could be correlated and scheduled.

The California concept of rating, very briefly, is this: As the member of the body disabled is of relatively greater or less importance in the occupation of the injured employee, the rating is accordingly increased or decreased from the standard. Thus, a structural steel worker would receive a higher rating for disability of a leg than would an office worker with a similar disability.

With reference to the age factor, disability increases with age. Thus, a young man with a given disability would receive a smaller rating than an older man with the same disability, assuming they were both in the same occupation. This is because a younger man has greater adaptability than an older man and usually would adjust more quickly to his handicap than would the more aged employee. Also, it is more difficult for the older man to obtain new employment. To reverse the principle so that disability rating decreases with age on the assumption that the younger man would have to carry his handicap for a longer period of time would be to permit the damage concept to enter into the disability evaluation. The occupation and age features

Presented before the Section on Industrial Medicine and Surgery at the 83rd Annual Session of the California Medical Association, May 9-13, 1954.

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do not add to the employer's cost. It simply means a more realistic distribution of compensation benefits.

The 1914 schedule was amended only slightly during the following 35 years. In 1950 a revised schedule became effective embodying the same basic principles as the earlier schedule. The basic ratings for individual disabilities were reexamined and revised. Occupations were regrouped in the light of changing industrial procedures, and the format was changed to afford easier use of the schedule. The adoption of the 1950 schedule followed several years of study of rating procedures throughout the world, and analysis of schedules from all domestic and many foreign jurisdictions. The Commission was fortunate in having available to it Robert E. Haggard, who for over 30 years had been associated with the Rating Bureau, and whose vast experience and knowledge were invaluable to the study.

Briefly, the present schedule contains a list of some 300 disabilities, each with a standard rating bearing a percentage of total permanent disability. The disabilities listed relate to the end result and not to the initial diagnosis. (Loss of motion of a wrist joint is listed, but a Colles fracture is not.) These disabilities are correlated with some 1,800 occupational titles, each of which is assigned to one of 60 occupational groups based upon the similarity of the physical demands of the occupations. A table for the age variant is included.

The percentage ratings specified are considered adequate on the average to compensate for the residual disability resulting from the injury and to afford a reasonable period of adjustment to the effect of such injury.

The California system has not been without criticism from both within and without the state. Most of the criticism is based upon the contention that the schedule is too complex. Other quarters question the validity of scheduling occupation and age. In reply to the first objection, the author would suggest that it is primarily the disability itself that is complex, and not the schedule. The schedule appears complex because it contains either listed disabilities or rules for rating many disabilities which would be nonscheduled under other types of schedules. Frequently those who object to a complex schedule at the same time object to nonscheduled ratings. It is obviously impossible to schedule all conceivable disabilities. Some degree of discretion is inevitable.

With reference to the second objection, it might be observed that it is far better to schedule occupation and age factors than to leave consideration of these elements to persons who have no common meeting ground for appraisal and whose personal views would lead to divergent conclusions.

The Labor Code specifies that four disabilities are conclusively presumed to be total in character. They are: loss of both eyes or the sight thereof, loss of both hands or the use thereof, an injury resulting in a practically total paralysis and an injury to the brain resulting in incurable imbecility or insanity. In all other cases total permanent disability must be determined in accordance with the individual circumstances in each case. The Labor Code further requires that in determining the degree of permanent disability consideration be given to the diminished ability of the injured employee to compete in an open labor market.

It is known from common experience that a blind man or a paraplegic man can earn. Therefore even the statutory total permanent disabilities do not necessarily contemplate that the injured employee is unable to work or is unable to earn money. Under the code it would appear that mere loss of ability to compete in an open labor market with other employees is sufficient to warrant a rating of total permanent disability. The employee need not be helpless and unable to do anything to earn a living.

This brings out the distinction between the purpose of temporary disability payments and of permanent disability payments. Temporary disability payments are based solely upon loss of wages during the healing period. Unless there is actual wage loss, there is no basis for payment of temporary disability, no matter how gravely injured the workman may be or how extensive his injury. The purpose of temporary disability compensation payments is to cushion the effects of actual wage loss incurred by the employee during the period of treatment and healing.

As to permanent disability payments, however, the concept is entirely different. They are based upon a prospective loss or impairment of earning power. This principle was illustrated some years ago by Mr. Gustav Michelbacher, who was chairman of the study which led to the formulation of the 1914 schedule:

"It is a matter of common knowledge that the laborer who, for example, loses one eye, while he may suffer no loss of earning capacity by reason of the physical impairment does suffer a loss in competing power, which is an important factor in determining the effect of this accident upon his future earning capacity. The worker who has lost an eye must compete for the rest of his life with healthy two-eyed workers, and even though he be physically able to perform the work equally as well as before the accident, he will still have difficulty in obtaining a chance to perform work in competition with other workers who are physically perfect."

The greater the permanent disability the longer the payments continue. This affords a longer period of rehabilitation and readjustment for the more serious disabilities, and a shorter period for the less

serious. In this connection Mr. William Leslie, who also participated in the creation of the 1914 schedule, wrote: "It is frequently referred to as the rehabilitation theory and assumes that the permanently injured worker either can or cannot regain his earning capacity. If he cannot, he must be compensated for life. If he can, he must be aided financially during the period of rehabilitation. The period of rehabilitation will on the average vary with the extent of disability."

Once the degree of permanent disability has been determined, payments are made under a formula set forth in the Labor Code, calling for four weeks of compensation payments for each 1 per cent of disability, and further providing that if the disability is 70 per cent or greater, there is in addition provided a life pension at a lower rate than the normal compensation.

In analyzing ratings and awards of the Commission, attention should be directed to the percentage of disability and not to the amount of money which the injured employee is to receive. The schedule provides a standard rating of 30 per cent of disability for the enucleation of one eye. This calls for 120 weeks of disability payments. The amount of the weekly payment varies according to the earnings at time of injury, from a minimum of $9.75 a week to a maximum of $30. Thus, the amount of money received could vary from a minimum of $1,170 to a maximum of $3,600. The important thing, however, is that regardless of the size of payments, they continue for the same length of time, in this instance 120 weeks.

From a procedural point of view, it should be remembered that in a controverted case it is the function of the trial referee to determine the factors of permanent disability that have resulted from the injury, on the basis of the record before him. Once these have been identified to the rating bureau, the factors presented are applied to the schedule and a recommendation is made to the referee. The report of the rating bureau is a recommendation only, and is not binding on the referee or the Commission. For that matter, the Labor Code provides that the schedule itself is but prima facie evidence and hence can be rebutted.

In informal or advisory ratings in which there is no formal proceeding before the Commission, the factors used as a basis for recommendation are extracted from the medical reports.

Now a physician might say, "This is all very well and good, but how do I fit into the picture? Of what use are my reports and how are they applied in a practical way?"

Medical reports are the keystone of awards of the Commission. They are as important to the Commission as a blueprint is to a construction engineer. Depending upon the particular question involved, the type of report desired may vary. In some cases the physician may be asked whether the alleged incident produced the disability claimed, in other cases, whether the asserted incident alone contributed to the end result, or whether preexisting or coexisting disabilities also contributed.

The author's remarks, however, are directed solely to reports pertaining to the permanent residuals of an accepted injury.

A clinical report may supply answers to etiologic questions and hence assist in identifying or denying liability in the first instance, yet may be of little help in the evaluation of a case from a rating viewpoint. The physician's report on factors of permanent disability is different in scope and character from the report which may have identified injury with employment.

It is essential that rating reports convey a thorough and accurate picture of the disability under consideration. The attending and examining physician therefore takes on a dual role—first, that of directing his professional skill and ability to curing and relieving the effects of injury; and second, that of being in a sense a reporter describing in clear and unmistakable terms the disability present. If a report is not clear it can cause needless misunderstanding and delay.

In a case involving industrial disability, the physician's report may pass through many hands during the adjustment of the case. The claims adjuster and perhaps the carrier's attorney, the applicant and perhaps the applicant's attorney, other examining physicians, as well as the staff of the Commission may all at some time review the report. All of these persons rely upon the reporting physician for an accurate picture of the disability involved in the case, since many of the persons who review the report never see the injured employee.

In an effort to eliminate misunderstanding and to afford physicians an outline of the information desired in the reports for rating, a committee composed of representatives of the California Medical Association and of the Commission, under the able chairmanship of Dr. Packard Thurber, submitted a report for the Standardization of Joint Measurements, some four years ago. The report was adopted by the Commission at that time and has since been published under the title of "Evaluation of Industrial Disability." Use of the procedures described is widespread but not universal, and the Commission earnestly requests that all physicians prepare their reports in accordance with the standardized procedure.

Disabilities such as those of amputation and lim-

ited motion are not difficult to evaluate, and frequently can be related directly to the Schedule. The nondemonstrable factors such as pain, weakness and sensory alterations pose the greatest problem to persons associated with ratings. The courts have found that pain, for instance, is ratable if it contributes to loss of earning power. Therefore if pain is present it must be included as a factor in the rating.

Only by a thorough description of such factors and their effect upon normal activities can the disability be adequately portrayed and rated. It is not sufficient to know that a man may have a slight, moderate or severe pain in a certain portion of the body. It is necessary that the persons determining the rating know the activities that produce the pain, the means necessary for relief, the duration of the pain and the activities that are handicapped or precluded by the pain. In short, it is necessary to know what the injured person can do despite the injury, what he can not do as a result of the injury and the extent to which disability is influenced by nondemonstrable factors.

The framework of the description is not much different from the "who, what, where, when and why" that journalists use in writing newspaper stories. The terminal report should not be directed solely to the site of injury but should include any secondary effects as well. For example, an ankle injury may result in limited ankle motion but also may produce permanent atrophy of the thigh muscles. The entire extremity should therefore be examined and a report made upon it.

With a complete picture the Commission and its staff are in a far better position to gauge the extent of disability and to make the proper and appropriate award. Obtaining the picture depends greatly on full and precise reporting by physicians. With understanding and cooperation, the joint task of physician and Commission will be made easier and the results more satisfactory.

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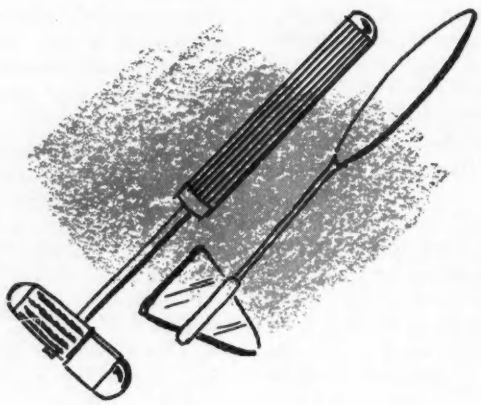

Congenital Absence of the Vagina

Surgical Correction

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• *In the two cases of congenital absence of the vagina reported herein, the embryologic point of arrest of development of the mullerian ducts was identical. Both patients had a rudimentary uterus, and one had a fibroid tumor on the rudimentary uterus. As a part of operation to construct a vagina in such cases, exploratory pelvic laparotomy appears to be a desirable step in order to determine the status of the internal genital organs.*

The use of a split thickness skin graft sewn around a vaginal mold and inserted into the dissected vaginal space results in more rapid healing, less scar tissue and a vagina that is soft, pliable and normal in appearance, even to the extent of having rugal folds.

ABSENCE OF THE VAGINA in a well-developed woman, normal in all other respects, is almost incredible, although not extremely rare. The incidence of the condition is not known exactly, but in view of the increasing number of cases being reported, it is probably much higher than it was once believed to be. Estimates vary from one case in 5,000 births[7] to six cases in 500,000 hospital admissions.[25]

As a rule the defect is not recognized until the age of puberty, for the external genitalia usually appear more or less normal. In many instances it does not come to notice until marriage is contemplated. Frequently the condition is associated with other anomalies of the internal genital organs, the most common of which is aplasia or rudimentary uterus with no endometrium and no tendency toward the development of hematometra. If the ovaries are present the patient is endocrinologically normal, but unless a vaginal canal can be constructed she cannot have normal sexual relations. In many instances neurosis develops from the patient's attitude toward this inadequacy. In several cases of record the urethra was sufficiently dilated to permit sexual intercourse.

The psychologic consideration is of great importance. Assurance that the condition is not very rare and that it will not, of itself, interfere with normal health will do much to help patients in whom the condition exists. Operative intervention should be postponed, if possible, until marriage is contemplated. Many surgeons believe that surgical correction of the defect should never be advised. However, when a patient with this condition wishes to be married or for other reasons wants the condition corrected, no amount of advice will change her mind.

EMBRYOLOGY

It is from the mullerian ducts that the entire internal genital tract of females develops. From the fifth to the sixth week of gestation, with the development of the genital glands on the mesial side of the wolffian bodies, the mullerian ducts develop on the lateral aspect of the wolffian ducts as a tubular invagination of the cells lining the coelum. The orifice of the invagination remains patent and undergoes enlargement and modification to form the abdominal ostium of the uterine tube. The mullerian ducts pass backward lateral to the wolffian ducts; but toward the posterior end of the embryo they cross to the mesial side of these ducts and end in an epithelial eminence, the mullerian eminence on the central part of the cloaca, between the orifices of the wolffian ducts. Later they open into the cloaca at this situation.

From the mullerian ducts develops the entire internal female generative tract to the vaginal introitus. The mullerian ducts are at first solid and extend to the urogenital sinus as separate, unfused structures. The fusion of the lower half of the mullerian ducts is complete in the ninth week of fetal life, the vagina still being solid but the uterus composed of two hollow compartments. The septum of the fused walls forming the uterus disappears from below upward. This part of the canal is lined by a single layer of epithelium and has two hollow compartments, whereas the vagina is still solid, being composed of large cells rich in protoplasm. The uterine musculature develops about the fourth month.

In the fourth to the fifth month, the uterus and vagina acquire a single communicating lumen. The uterus and vagina are distinguished from each other by the formation of the cervix at the beginning of the fifth month. The hymen appears in the fifth month as a special differentiation of the lower vaginal segment and represents the remains of the mullerian eminence.

From the College of Medical Evangelists, Los Angeles 33.
Submitted April 6, 1954.

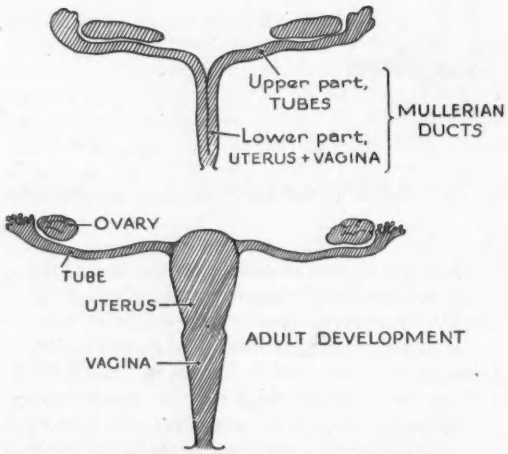

Figure 1.—Diagram of embryologic development of mullerian ducts resulting in adult internal genital organs: tubes, uterus and vagina.

Figure 1 is a diagrammatic representation of the transformation of the embryologic mullerian ducts to the adult stage of development.

METHODS OF CONSTRUCTING AN ARTIFICIAL VAGINA

Many different methods have been used for the construction of an artificial vagina, with varying degrees of success.

Rectal Transplant. Sneguireff[26] in 1892 and Gersuny[14] in 1897 apparently first utilized a segment of bowel to create an artificial vagina. Part of the coccyx was removed, 12 inches of the rectum was transposed forward to the new opening for the vagina, and the upper limb of the rectum was drawn down through the rectal sphincter.

Ileal Transplant. Baldwin[2] in 1904 described a method by which a single or double loop of bowel was brought down to the perineum to form a vagina and an end-to-end intestinal anastomosis was done to restore intestinal continuity.

There has been a definite trend away from the bowel operations toward simpler methods because of the high mortality rates reported by Masson[21] and Novals.[24]

Labial Grafts. Graves[16] in 1908 revived interest in the labial flap operation. Grafts were fashioned from the labia and sewed around a vaginal mold which was then inserted into the dissected vaginal space. There have been several modifications of this technique.[6, 8]

Thigh Graft. Beck[3] in 1900 developed the vaginal space and inserted a tube graft made from pedicle flaps from the thigh. Frank and Geist[11] modified this method and used a multiple stage procedure with full thickness grafts. Grad[15] used a similar technique in two stages.

Simple Pressure. Frank[12, 13] devised a method of creating an artificial vagina, without operation, by daily pushing of graduated tubes against the perineum. This would be an ideal method if sufficient depth could be obtained in all cases and if the patients would be completely cooperative.

Simple Reconstruction. This technique consists of merely opening the vaginal space, inserting a vaginal mold and waiting for the tract to epithelialize itself without the use of a skin graft. Wills,[27] Kanter[19] and Wharton[28] reported good results with this technique.

Wharton expressed belief that the epithelium from the urogenital sinus, forming the lowermost part of the vagina, will grow to form the artificially created vagina. However, Counsellor[5] reported cases in which this did not occur in a period of two years and grafting was required.

Free Skin Grafts. Free skin grafts were first used in 1872 by Hepper[17] and have since been reported by Flynn,[10] and Kirschner and Wagner.[20]

McIndoe and Banister[22] in 1938 reported the use of a Thiersch graft over a vulcanite mold which was left in place three to six months. They stressed the prolonged use of the mold to overcome the "contraction factor" of the new tract. Counsellor[5] reported good results with this method in 1944. Although Wharton[28] originally felt that skin grafting was unnecessary, later he favored the use of a Thiersch graft over the mold to hasten epithelization and to decrease the amount of scar formation.[29]

Miller, Willson and Collins[23] reported 17 cases. In six reconstruction was done without graft and in 11 the graft was used. They felt that the use of graft gave better results because the vagina was softer, more pliable and better epithelized than when a graft was not used. Ferris[9] also felt that a graft should be used wherever possible.

OPERATIVE PROCEDURE

Preoperative preparation should always include a study of the upper urinary tract. Because of the embryologic association of the genital and urinary tracts, congenital absence of the vagina is frequently accompanied by urinary anomalies such as pelvic kidney, solitary kidney and duplicated ureters.

The surgical technique used in the cases reported herein consisted of three steps: (1) Abdominal pelvic exploration. (2) Split thickness graft, taken from the thigh, sewed around a vaginal mold. (3) Dissection of the vaginal space, careful hemostasis and the insertion of the graft covered vaginal mold.

Abdominal pelvic exploration is done to evaluate the upper genital tract and accurately determine sex. Horn[18] raised these pertinent questions: Does the patient have normal fusion of the upper mullerian apparatus? Does she have a normal sized uterus

and is it functional or could it be made to function? (If so, it should be connected with the vagina to be constructed.) Is the patient really a female, or a male with female breasts? (Cases of male pseudohermaphroditism, i.e., males with normal appearing female breasts, have been described.[4]) Or is this a case of true hermaphroditism? In order to completely and finally answer these questions of vast personal importance to the patient, abdominal exploration is recommended.

There seems to be general agreement as to the satisfactory results obtained by dissection of the space between the bladder and rectum and the maintenance of this space by the insertion of a Pyrex glass mold of the proper size. Some investigators have expressed belief that epithelium will grow up around the mold from the vulva in a short time, whereas others feel that a graft will give better results. Owens[25] stated that in light of the difficulty encountered in attempting to epithelize completely a newly formed vaginal cavity by means of a split thickness skin graft, it is not easy to understand the facility with which the entire cavity was epithelized solely by the proliferation of vaginal mucosa. Counsellor said that where epithelization does not occur in two years, grafting should be done. In the cases herein reported, grafts were used and the vagina was almost completely healed in two months. In addition, the resultant vagina was free from scarring, was soft and pliable. Fortunately, even rugal folds in the vagina were produced.

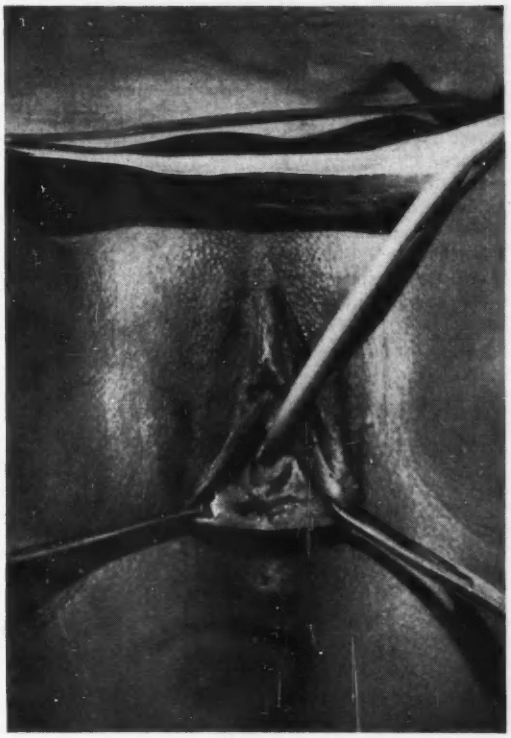

Figure 2.—Congenital absence of the vagina (Case 1) with normal clitoris, labia majora and minora and urethra.

CASE REPORTS

CASE 1. A 25-year-old white woman who had never menstruated was having sexual difficulties after one month of marriage.

Although aware that not to have menses was abnormal, she had not consulted a physician but had hoped that menstruation would "some day start." The patient said she had had mild lower abdominal pains intermittently.

The body contours, breasts and distribution of hair were normal. The clitoris, the labia majora and minora and the urethra were also normal (Figure 2). No vaginal introitus or dimple could be found on careful inspection of the mucous membrane from urethra to anus. The mucosa could be forcibly invaginated a distance or no more than 1 cm. Upon rectal examination a mass 3 cm. in diameter was palpated. It was thought this might be an infantile, nonfunctioning uterus. An intravenous pyelogram showed the upper and lower urinary tracts to be normal.

A midline suprapubic incision was made and the peritoneum was opened. The bladder, rectum, tubes and ovaries were normal, as were the round, suspensory (infundibulopelvic) and uterosacral ligaments. The tubes had normal fimbriated ends laterally, but medially they ended in a "nubbin" of tissue 2 x 3 cm. in diameter on each side about 5 cm. apart (Figure 3).

With the abdomen open, the perineal part of the operation was done by making a transverse incision between the urethra and the rectum, after inserting a Foley catheter (see Figure 3). The space between the urethra and rectum was dissected carefully by a combination of sharp and blunt dissection until the peritoneum (identified by the hand of an assistant reaching into the peritoneal cavity and pressing from above) was reached. The abdominal incision was later closed in layers. A small amount of bleeding was controlled by pressure. The space was gradually enlarged to admit two, then three and finally four fingers; and then the vaginal mold was inserted.

With a Brown electrodermatome a split thickness skin graft was taken from the inner aspect of the thigh. The mold was removed, the graft was sewn around it "inside out," and then the graft and the mold were inserted and held in place by two heavy silk sutures to the labia (Figure 3).

There was no fever after operation. In order not to disturb the graft the patient was kept flat in bed for two weeks. The large vaginal mold produced pressure, and consequently difficulty in urination and bowel movements. (Cases have been reported

elsewhere in which the mold eroded into the rectum.) The Foley catheter was removed after one week and subsequently the patient urinated spontaneously. A liquid diet was given during the first week and solid foods the second. During the second week the bowels moved spontaneously. The sutures in the mold were replaced by strings to keep the mold in place. With the graft healing nicely, the patient was permitted to go home on the sixteenth postoperative day.

Weekly examination was done in the office and each time the mold was removed and sterilized. The graft was inspected and granulating areas were touched with silver nitrate.

At the end of two months healing was complete and there was a normal appearing vaginal introitus (Figure 4). The depth and width were normal also. Biopsy of tissue from the vaginal vault in the third month showed the lining to be normal epithelium with practically no inflammatory reaction (Figure 5).

The mold was kept in place almost continuously for seven months. The patient was permitted to have sexual intercourse in the fourth postoperative month. Sex relations were reported to be satisfactory for herself and her husband.

CASE 2. A 30-year-old white woman who had been married one week said that sexual relations were unsatisfactory and that her husband, attempting coitus had injured her bladder and produced blood in the urine. She had gone to her family doctor who told her she did not have a vagina. She had been treated five years previously by the author for an unrelated minor surgical condition and had been told then that she did not have a vagina. Other physicians also had told her of the defect, but she put the problem from her mind until she married. Upon physical examination normal secondary sex characteristics were noted, and all external genitalia were normal except for absence of the vagina. An intravenous pyelogram was normal.

Construction of an artificial vagina and exploratory laparatomy were done by the technique used in Case 1. The pelvic findings were identical with those in Case 1—normal tubes and ovaries. In order to rule out true or pseudohermaphroditism, a biopsy of the gonad was taken. The report was "normal

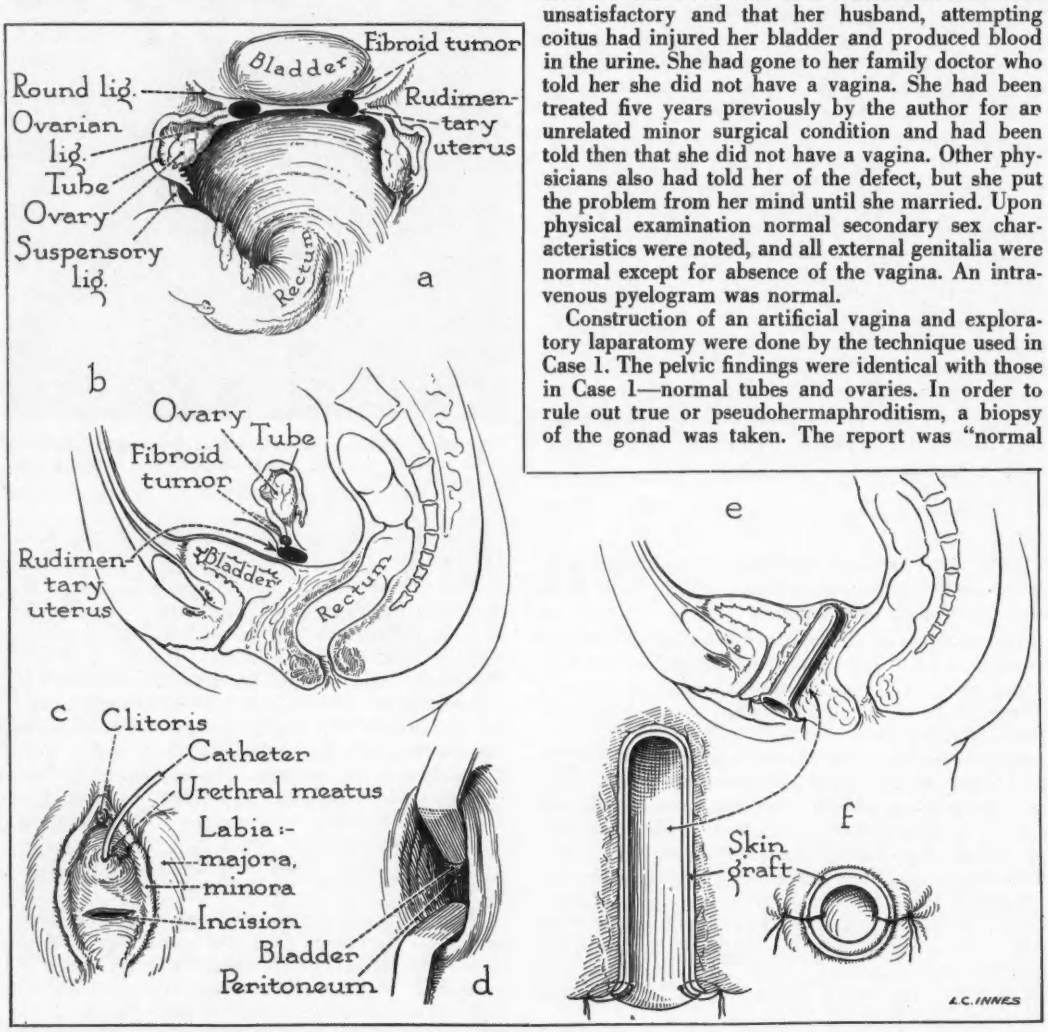

Figure 3.—Diagram showing (a) pelvic findings in both cases herein reported: Normal bladder, rectum, tubes, ovaries, round and suspensory ligaments. In both cases tubes ended in "nubbins" of tissue which proved to be rudimentary, unfused, uterus. The condition in Case 2 is shown on the right side, demonstrating a fibroid tumor on the right rudimentary uterus. (b) Midsaggital section showing absence of any evidence of a vagina. (c) Operative procedure—transverse incision made and dissection to be continued between urethra and bladder anteriorly and rectum posteriorly. (d) Dissection carried to the peritoneum. (e and f) Skin graft sewn around vaginal mold and inserted into vaginal space. Mold sutured to labia.

ovary with corpus luteum." The tubes, as in Case 1, ended in a "nubbin" of tissue, which was thought to be a rudimentary, unfused uterus. On the "nubbin" on the right side there was a nodule 0.5 cm. in diameter (Figure 3). Biopsy of tissue from the area showed the nodule to be a fibroid tumor on a rudimentary uterus.

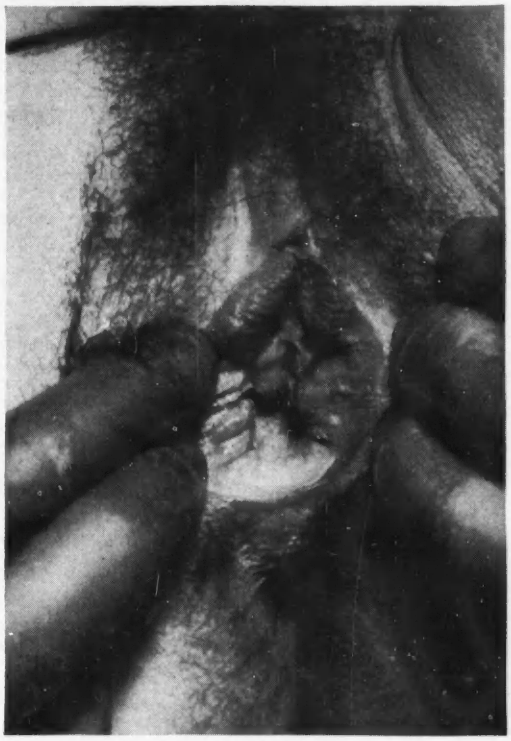

Figure 4.—Photograph (Case 1) taken two months after operation, showing normal-appearing vaginal introitus.

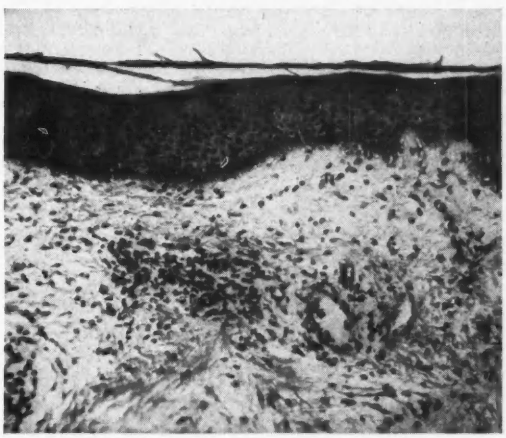

Figure 5.—Slide of biopsy specimen (Case 1) taken from vaginal vault in third postoperative month, showing lining of normal epithelium with practically no inflammatory reaction.

The postoperative course was as described under Case 1. Healing was complete at the end of two months and the vagina was of normal depth and width. The texture was practically normal, even to the presence of rugal folds (Figure 6).

Sexual intercourse was permitted in the third month and the patient reported completely satisfactory sexual relations both for herself and her husband, with orgasm.

DISCUSSION

In both the cases here presented, the stage at which arrest occurred was the same—failure of development of the lower half of the mullerian ducts. The mullerian ducts never reached the midline and the mesenchymal tissue therefore condensed about the medial ends of the tubes, resulting in "nubbins" of normal myometrial tissue—so normal, in fact, that in Case 2 a fibroid tumor had already started to grow.

There can be little doubt that had the mullerian ducts progressed just a little bit farther—until they had met in the midline—a uterus might have developed in each case. In fact, there are reports of cases in which, when the vagina was constructed, communication with the uterus was established and pregnancy resulted.[1, 30]

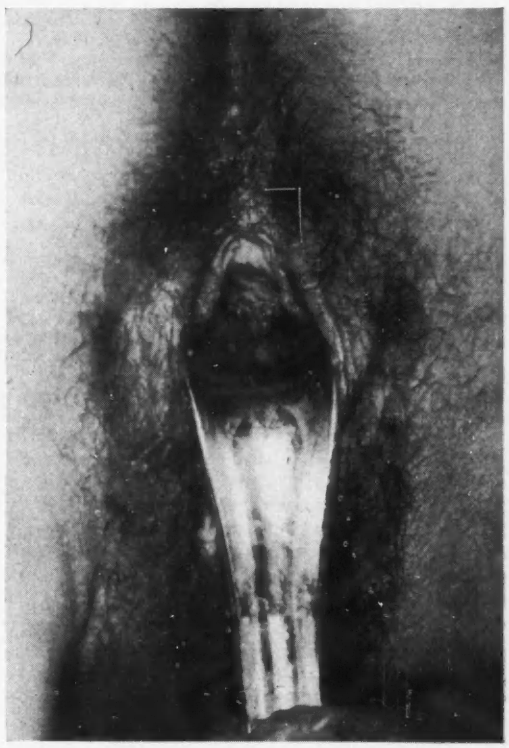

Figure 6.—View of posterior vaginal wall showing normal texture, even to the presence of rugal folds.

Since the stage of arrest of development of the mullerian ducts was identical in the two cases reported herein, the literature was reviewed on that point specifically. Six reports[31] of cases in which developmental arrest occurred at this same point were found. How often this occurs is unknown, for many of the articles did not specify and in many cases exploration was not carried out. But it is evident that this particular point of arrest must not be uncommon, which gives cause to wonder what might be the underlying mechanism.

ADDENDUM

Recently another patient was observed who had been operated on elsewhere for construction of a vagina. Urethrovaginal and rectovaginal fistulae resulted. The patient was in the hospital for three months. When the fistulae healed, the constructed vagina was badly scarred. This case is cited to illustrate the problems of keeping in the proper plane and the complications and poor results that may ensue.

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CASE REPORTS

Severe Hypotensive Reaction to Oral Chlorpromazine Therapy

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SINCE 1951 Thorazine® (chlorpromazine) has been experimentally studied and extensively used clinically.[6, 7] It has been reported extremely effective in preventing or controlling nausea and vomiting in many conditions,[1, 3, 4, 5, 10] in relieving various psychic disorders through tranquilizing effects[2, 10, 11] and for diminishing pain.[10, 11]

Certain undesirable but relatively inconsequential and transient reactions have been noted clinically—drowsiness, minor gastrointestinal disturbances, dryness of the mouth, dizziness, tachycardia and headache. Other effects of greater concern have been severe and prolonged jaundice of obstructive type and hypotension. Neither has resulted in a reported fatality, but the authors recently observed a patient who had prolonged and gravely severe hypotensive reaction from four 25 mg. doses of this drug by mouth.

Moyer and associates[8, 9] observed moderate hypotensive responses to Thorazine in dogs in parenteral doses of 0.1 to 0.5 mg. per kilogram of body weight. There was a uniform severe fall in blood pressure in doses of 10 mg. per kilogram, presumably largely due to peripheral vasodilatation, but likewise associated with decreased cardiac output. Atropine did not block this reaction. In the clinical cases studied there was a 12 per cent incidence of hypotension after administration of Thorazine. In most patients the fall was mild and transient, lasting but a few minutes, but in seven patients the blood pressure fell by from 30 to 60 mm. of mercury after intramuscular administration. In three of these cases the pressure fell to 90/60 mg. of mercury. Other investigators reported similar reactions. Winkelman[11] reported a fall in blood pressure in seven of eight patients ranging from 5 to 60 mm. of mercury systolic, and from 5 to 25 mm. diastolic. The greatest decreases occurred in patients with hypertension, and the use of Thorazine as a therapeutic agent has been suggested for this condition. Occasional episodes of dizziness, tachycardia, postural hypotension and in some instances transient syncope have been reported. However, in all these reports spontaneous recovery occurred in from one-half to three hours with no serious or prolonged sequelae resembling severe clinical shock.

The following case is presented because it is believed to be an example of a critical shock-like state attributable to oral administration of Thorazine.

REPORT OF A CASE

A 71-year-old white male was admitted to hospital with complaints of fever, malaise, increased cough and hoarseness, anorexia, insomnia and psychic irritability of three days' duration. He had been under care for the six preceding years for emphysema and chronic and acute recurrent bronchitis. Four years previously he had had acute myocardial infarction, from which he recovered uneventfully. Three months before the present admission he had received roentgen therapy for squamous cell carcinoma of the esophagus and although clinically and roentgenographically the lesion was resolved, a preexisting productive cough was exaggerated. Five weeks before the present admittance the patient was hospitalized with bronchitis and pneumonitis of the left lower lobe, and a diagnosis of radiation-induced pulmonary fibrosis was also made. The blood pressure at that time ranged from 120/72 mm. to 130/78 mm. of mercury.

On the present admission similar diagnoses were made. The maximal oral temperature was 39.4° C. the day prior to admission. Upon physical examination musical bronchial rales were noted. The blood pressure was 96/70 mm. of mercury, the pulse rate 120 per minute, the temperature 38.2° C. and respirations 36 per minute. Mild hypochromic anemia was the only abnormality noted in laboratory tests. The patient was given 600,000 units of penicillin and 0.5 gm. of streptomycin intramuscularly twice daily, and Pyribenzamine,® 50 mg., four times daily by mouth. On the third day, erythromycin, 200 mg. twice daily was given orally, and the penicillin and streptomycin were discontinued. The daily rectal temperature fluctuated between 37° C. and 38.2° C. for the next six days. Crepitant rales in the left lower lobe developed on the second hospital day.

By the sixth hospital day, the patient had less malaise and cough, and the rales in the left lung cleared. During this period the blood pressure varied from 108 to 140 mm. of mercury systolic and 50 to 66 mm. diastolic. Anorexia, insomnia and psychic irritability persisted with increased restlessness. The patient refused meals and attention, and in an attempt to control the mental irritability, Thorazine

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Submitted November 1, 1954.

was given orally in a 25 mg. dose at 5:30 p.m. on the sixth hospital day. The patient slept well that night, and the next morning (the seventh hospital day) was less restless and had improved appetite. At 10 a.m. the blood pressure was 134/66 mm. of mercury. Thorazine was administered as follows: a 25 mg. tablet at 8:30 a.m., another at 12:30 p.m., and another at 5:30 p.m. that day. Twice during the day when the patient sat in a chair for about 15 minutes he complained of faintness, but the blood pressure was not determined at those times. About 2:30 p.m. he had a transient episode of tachycardia and tachypnea when sitting up, which was promptly relieved by reclining. The evening was spent quietly in bed, and at 9:00 p.m. the patient was comfortable with a pulse rate of 94 and respiratory rate of 36 per minute. At 9:30 p.m. with the patient semi-reclining in bed the blood pressure was 104/60 mm. of mercury but at 9:50 p.m. he complained of weakness and the blood pressure was 74/40 mm. Respirations were 40 per minute and the pulse rate 130 per minute. The patient's color and skin temperature remained good throughout, and the only other complaint was of tingling in the hands. Five milligrams of Neo-synephrine was given intravenously, and in 10 minutes the systolic blood pressure rose to 180 mm. of mercury; after another ten-minute interval it was 85 mm. Oxygen was given nasally and l-norepinephrine therapy by intravenous drip was started. An electrocardiogram taken at this time showed sinus rhythm with a rate of 100, and minor T and S-T deviations from the normal not significantly different from those in a tracing taken three months earlier revealing the fixed residual findings resulting from the anterior myocardial infarction of 1950. The leukocyte content was 10,700 per cu. mm. of blood at 10:30 p.m. with normal differential.

For the next day and a half the systolic blood pressure was maintained between 92 and 110 mm. of mercury by intravenous administration of l-norepinephrine solution in a concentration of 4 mg. per liter at a rate of flow of 12 to 20 drops per minute. Any attempt to lower the dosage resulted in a fall of systolic blood pressure to between 70 and 80 mm. of mercury. An electrocardiogram taken on the seventh day showed the cardiac rate at 89 per minute but no other changes. At 4 p.m. of the seventh hospital day the patient was erroneously given an additional 25 mg. tablet of Thorazine by mouth. At 4:30 p.m. there was a decrease in blood pressure to about 70/50 mm. of mercury and for three hours when weaning from l-norepinephrine was attempted the blood pressure would fall to this level. In about four hours the rate of drip of the l-norepinephrine solution (4 mg. per liter) necessary to maintain systolic blood pressure over 90 mm. of mercury had to be increased from 10 drops per minute to 42 drops per minute. Concentration of l-norepinephrine was then increased to 8 mg. per liter, permitting reduction of the rate of drip required to maintain systolic blood pressure between 90 and 110 mm. to 20 drops per minute. Since the hypotensive state had persisted 24 hours and for 6 hours after the accidental dose of Thorazine, 200 mg. of hydrocortisone was given by mouth in an attempt to sensitize the vasopressor system. Four hours later an additional 100 mg. of hydrocortisone was given by mouth, and then 25 mg. at four-hour intervals for four doses daily. Within 12 hours of the start of hydrocortisone therapy the blood pressure was stabilized at about 120/70 mm. of mercury without the aid of l-norepinephrine, and remained at about that level. Hydrocortisone was continued at 25 mg. orally in three doses daily and the patient was discharged from the hospital four days later free of symptoms except for a mild cough.

An electrocardiogram taken 12 days after admission showed sinus rhythm, a rate of 100 per minute, occasional ventricular premature beats and the fixed residual findings of the anterior myocardial infarction of four years before but recovery from the findings (on the sixth hospital day) of the depression of S-T segments which had suggested transient subendocardial hypoxia. There was no other significant change from previous records. At no time was there pain in the chest, dyspnea, peripheral edema or pulmonary edema. No pericardial rub or changes in cardiac sounds were heard. After cessation of all medications except hydrocortisone and auxiliary potassium in daily doses of 4 gm., the patient did exceptionally well and was discharged on the twelfth hospital day.

COMMENT

A case is reported of severe and prolonged hypotension following four 25 mg. oral doses of Thorazine. Administration of l-norepinephrine by vein for a day and a half was required to maintain effective arterial blood pressure. Intensive oral hydrocortisone therapy in addition may have contributed to the patient's recovery. The accidental administration of an additional oral dose of 25 mg. of Thorazine given 18 hours after onset of hypotension seemed to intensify the hypotensive reaction.

ADDENDUM

Since the above report was prepared, an article has appeared summarizing seven more cases of severe hypotension due to Thorazine,* including one in which the patient died.

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Psychosomatic Leukorrhea

Report of a Case

MILTON O. KEPLER, M.D., Fresno

VAGINAL LEUKORRHEA is a common gynecologic complaint. Common causes include infections of the vaginal tract, such as those caused by *Tr. vaginalis*, *Monilia albicans*, and *N. gonorrhea*, and inflammations of the cervical and endocervical areas. Neoplasms must be considered. Psychic causes, however, do not seem to be commonly listed among etiologic factors. It is well known that erotic stimulation can cause leukorrhea of at least a temporary nature, and it follows that the psyche may cause this condition. Following is a report of a case of persistent leukorrhea, caused by psychic conflict, which almost escaped detection because of several complicating factors.

REPORT OF A CASE

The patient, a 28-year-old, white, married woman was first observed for a routine prenatal examination. She had been married for about two years and the pregnancy was her first. She said that another physician had told her she had a "contracted pelvis" and that she might have to undergo uterine section for delivery. She added that the physician also had treated her for a troublesome vaginal discharge and that nothing he prescribed had given relief. The discharge was described as thick, white, non-irritating, not foul, but copious enough to necessitate wearing a pad.

She said that she and her husband were compatible in all respects and that intercourse was satisfactory. Throughout the history, examination and subsequent visits, the patient always maintained an air of sophistication coupled with complete candor in discussing matters of sex.

The patient was in about the seventh month of gestation. The pelvic outlet was moderately narrowed in the transverse diameter. The vaginal vault contained an excessive amount of mucus, but no cause for this was found except the pregnancy. A hanging drop preparation was negative for *Tr. vaginalis*. No pathogenic organisms were seen on examination of smears of material from the urethra.

The patient was advised that uterine section probably would not be necessary, since the fetus was small, and that the vaginal discharge probably was physiologic, even though it was more copious than usual. She was instructed, however, to use Floraquin® suppositories nightly in an effort to reduce the secretions.

At the next visit two weeks later the patient was distressed because the discharge had not lessened. She said that coitus had been rather painful for her lately, and that the vagina seemed to be rather "small," with actual friction causing the pain. Her husband, she added, was very considerate and always took time enough to stimulate her prior to the conjugal act. Instructions were given the patient to refrain from further contact insofar as intercourse, douches, etc., were concerned, since the time of delivery was so near. Just before time for the patient's next visit, the patient's husband telephoned that his wife was having considerable gastric distress. Upon examination it was found that the distress was in fact labor. The patient was admitted to hospital and, with midline episiotomy, was quickly delivered of a viable male baby weighing 5 pounds 6 ounces and estimated to be a month short of term. Mother and baby did well during the hospital course and the puerperium was uneventful. At examination six weeks after delivery, involution seemed complete, with good healing at the site of episiotomy. Some Nabothian cysts of the cervix were noted but they were not treated at the time. The patient continued to complain of leukorrhea and occasional dyspareunia but no physical cause could be found. On a subsequent visit cautery was used to treat the cysts, and the endocervix was cauterized by the bi-active coagulation electrode technique on three occasions. A hemorrhage occurred from the site of one of the cauterized cysts about ten days later and additional cauterization was necessary to stop it. Normal menstruation was soon resumed but the patient still complained of leukorrhea and moderate dyspareunia. Acid douches and antiseptic suppositories did not help, nor did the use of lubricating jelly.

With the patient's consent, the husband was interviewed. He was pleasant and intelligent but, concerned over his wife's condition, he considered sending her to a medical center for further study and treatment. Encouraged to speak of his wife and her background, he said that she had begun to have leukorrhea the day they were married, even though, because of moving and other circumstances, they had not had intercourse until a week later. He had never felt that his wife had enjoyed the act, even though her libido seemed adequate; he doubted that she had had more than a few orgasms during the course of their marriage. She was of a typical middle class family of "working people," never in want and never in luxury. The parents were good enough to the

Submitted May 29, 1954.

daughter, but they did not share intimacies or discuss matters relating to sex, nor did they encourage the girl to develop normal relationships with boys. There were no brothers. Dates were infrequent; she had not gone out with men at all until after graduation from high school. She had been frightened by some of the advances that were made to her by various men, and her husband felt that she was very naive regarding men and the actual mechanics of sex.

The husband was asked to have a thorough talk with his wife and to send her back for additional talks with the author. Subsequently all the facts related by the husband were confirmed. Asked why she had not spoken of these matters before, she merely said that she had not considered the facts important enough to tell. In the course of a few talks, she gained insight into her problem—that she had actually feared sex and the sex act, and that leukorrhea was actually a defense against it. Care was taken not to offer this explanation to the patient until she herself suggested it. Leukorrhea thereupon dramatically ceased, as did dyspareunia.

COMMENT

At the time the present report was being written, the author conferred with various physicians regarding psychosomatic leukorrhea. Most gynecologists consulted agreed that it was a common condition but spontaneously stated that the cause was often not discovered. It would seem, then, that psychic factors should always be considered in the complaint of leukorrhea.

4771 East Grant Avenue, Fresno.

Nontuberculous Giant Lung Abscess Complicating Tuberculous Bronchostenosis

JOSEPH GOORWITCH, M.D., Los Angeles

A TUBERCULOUS CAVITY occupying most of or an entire pulmonary lobe is not a very rare phenomenon; neither is a nonspecific abscess of similar dimensions. However, a giant abscess occupying the entire upper lobe and developing subsequent to and beneath an adequate thoracoplasty is uncommon, especially when unaccompanied by either symptoms, signs or x-ray findings of an abscess. Such a case constitutes the subject of this report.

CASE REPORT

The patient, a 34-year-old woman, had had pulmonary tuberculosis diagnosed at the age of 18 in the course of a routine survey at school in 1937. Upon x-ray examination minimal infiltration was noted in both upper lobes. During residence of the patient in sanatoria from 1937 to 1949 no definite evidence of cavitation was found; except for the formation of a dense nodular lesion at the level of the left second anterior intercostal space, there was progressive clearing of the parenchymal lesions in both lungs as late as 1941. Left phrenic crush was performed in 1939 and an anatomically inadequate pneumothorax on the left was maintained from 1941 to 1943.

In 1942 symptoms of tuberculous tracheobronchitis were noted and sputum was found to be positive for tubercle bacilli for the first time. Repeated bronchoscopy in the course of the next few years revealed extensive tuberculous ulceration of the trachea and left bronchus which went on to healing by stenosis of the left stem bronchus. At the same time roentgenograms showed atelectasis of the left upper lobe (Figure 1, *left*). In 1948 three-stage, seven-rib left thoracoplasty was performed. Thereafter there was atelectasis of the entire lung (Figure 1, *right*) and the sputum remained positive on culture.

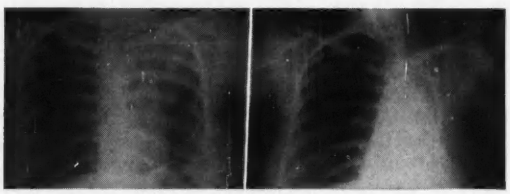

Figure 1.—*Left:* Roentgenogram (Nov. 24, 1947) showing atelectasis of the left upper lobe. *Right:* (Dec. 22, 1948) An anatomically satisfactory thoracoplasty but also atelectasis of the entire left lung.

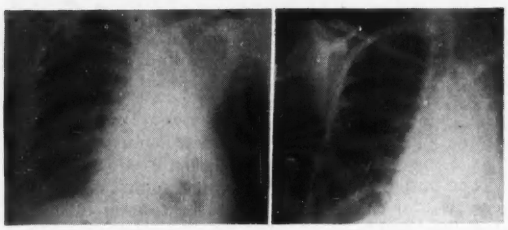

Figure 2.—Roentgenograms shortly before (*left*) and after (*right*) pneumonectomy, showing an almost identical appearance.

When the patient was first observed at the Mount Sinai Clinic in 1953, upon review of the roentgenograms no evidence was seen of tuberculous activity since the date of thoracoplasty in 1948 (Figure 2, *left*). At this time there was no growth on culture of three specimens of sputum. The patient said she had had episodes of low-grade fever, pains in the chest and increase in cough over the preceding three years without purulent sputum at any time.

A diagnosis of "destroyed" lung due to atelectasis and bronchiectasis resulting from healed broncho-

From the Department of Thoracic Surgery, Mount Sinai Hospital, Los Angeles.

Submitted April 12, 1954.

stenosis was made. Left pneumonectomy was carried out October 19, 1953, with the patient in the lateral position. In freeing the lung from its extensive parietal attachments it was noted that the lower lobe was shrunken but the upper was greatly distended and felt tense. In the course of manipulation the upper lobe was accidentally damaged and creamy pus escaped from it into the pleural cavity. It was considered wiser to evacuate the entire contents of the lobe (200 to 300 cc.) than to repair the tear in the lung and run the risk of flooding the tracheobronchial tree with liquid contents of the giant pulmonary abscess. At no time during the operation were pleural contents recovered from the trachea by the anesthetist, nor was there escape of air from the pulmonary abscess, which indicated complete isolation of the abscess from the tracheobronchial tree. The stem bronchus was extremely stenotic and required little in the way of repair. Postoperative recovery was uneventful (Figure 2, *right*).

Upon pathologic examination* of the surgical specimen (Figure 3) it was noted that the lower lobe was contracted and moderately bronchiectatic. The upper lobe consisted of a thin-walled trabeculated pus sac with only a very small area of tightly compressed pulmonary tissue near the hilum. The stem and upper lobe bronchi were extremely stenotic and one of the segmental branches was completely obstructed by old fibrosis.

It was noted that in sections through the site of bronchial occlusion the lumen was filled with connective tissue which in places was almost collagenous; there were only a few remnants of bronchial mucosa and glands. Degenerated tissue lined by fibrous wall was observed in sections through the wall of the cavity of the abscess. Sections through the lower lobe showed only pronounced atelectasis. No definite evidence of tuberculosis was found in any of the sectioned tissue. No pyogens or tubercle bacilli were observed on smears of the purulent contents of the abscess, and no tubercle bacilli grew on cultures of the material.

COMMENT

The interesting feature of the case here reported is the unexpected finding of a giant abscess occupying a completely excavated pulmonary lobe adequately collapsed by thoracoplasty five years earlier. Gradual accumulation of secretions distal to the completely obstructed upper lobe bronchus resulted in pressure that caused necrosis of pulmonary parenchyma with eventual conversion of the lobe into a pus sac. Regardless of whether or not this type of lung abscess can be diagnosed preoperatively, the fact that Lindskog and Liebow[1] put it last on a list of lesions associated with tuberculous bronchostenosis suggests that the incidence is rather low.

*By Irving Matoff, M.D.

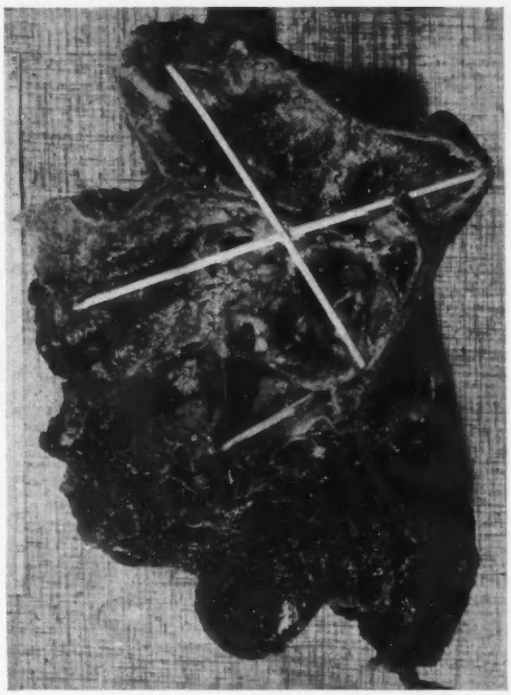

Figure 3.—Fixed gross specimen showing the inner aspect of the excavated upper lobe following evacuation of purulent contents and the compressed lower lobe. Bronchi are extremely stenotic.

SUMMARY

An uncommon nontuberculous complication of healed tuberculous bronchostenosis is reported in a patient who 16 years earlier had only minimal parenchymal disease but in whom extensive ulceration of the trachea and left bronchus developed later. Collapse therapy was carried out but the sputum remained positive for tubercle bacilli. A preoperative diagnosis of "destroyed" lung due to healed tuberculous bronchostenosis was made and pneumonectomy performed.

Unexpectedly the resected lung was found to consist of a completely excavated and distended upper lobe containing creamy pus and a shrunken moderately bronchiectatic lower lobe. The upper lobe bronchus was completely obstructed by the healed tuberculous process. Histologic and bacteriologic examination disclosed no evidence of tuberculosis in the bronchus, parenchyma or the purulent contents of the lung abscess.

1052 West Sixth Street, Los Angeles 17.

REFERENCE

1. Lindskog, G. E., and Liebow, A. A.: Thoracic Surgery and Related Pathology, 1st ed., Appleton-Century-Crofts, Inc., New York, 1953, pp. 200-202.

California MEDICINE

For information on preparation of manuscript, see advertising page 2

EDITORIAL

Allergy Skin Testing

THE SKIN TEST has been a most important functionary in the evolution of the subject of allergy. Since the development of the scratch and intracutaneous tests as diagnostic procedures in hereditary forms of hypersensitivity, allergists have come to appreciate their importance and their limitations. The skin test is, at best, a relatively crude diagnostic technique which, although simple to carry out, is subject to many pitfalls. Considerable experience is necessary for the appraisal of reactions obtained with it.

The scratch and the intracutaneous techniques are the methods most generally employed in testing for allergy of the hereditary type. The scratch test is performed by gently rubbing allergenic material into a superficial skin scratch. The intracutaneous test is done by introducing a previously calculated amount of allergen into the superficial layers of the skin. Both procedures have their limitations and advantages. The scratch technique is fairly simple and inexpensive and diminishes the likelihood of constitutional reactions that are liable to occur with careless and inexperienced use of the intracutaneous method. On the other hand the intracutaneous test is more sensitive than the scratch test and is, accordingly, a more effective diagnostic procedure. A decided advantage of the intracutaneous method is its adaptability and flexibility. The testing allergens may be varied in concentration to suit the need of each patient. The same materials used for testing may be employed also for desensitization treatment.

Many modifications of the scratch and the intracutaneous tests are being used. Some allergists recommend and employ the puncture test which is conducted by needle puncture through a drop of allergen. Others, as a variation of the scratch technique rotate the end of a fine narrow borer or screw driver on the skin and then rub allergen into the slightly abraded area. Another method is to mix the allergen with a vehicle that readily penetrates the skin and then gently rub it in. Still another and more novel technique is the introduction of an allergen into the skin by electrophoresis. However, in all these procedures the same immunologic principles apply, and the various techniques have been derived simply to satisfy the particular needs and preferences of the investigator. Most clinicians concentrate on one technique, and perfect it to a point where it provides for them more information than would be obtained with the less skillful application of several methods.

Many factors, nonspecific and immunologic, contribute stumbling blocks to a neophyte in the interpretation of the clinical significance of cutaneous reactions: the skin reactivity of each person differs from that of others; the skin reactivity differs from place to place on the skin of each person; the skin reactivity fluctuates with age and may be suppressed in certain diseases; the skin reactivity is influenced by the concentration and amount of allergen employed; the cutaneous reaction may result from different immunologic mechanisms in the same person and vary in clinical significance; and the cutaneous reactions in different persons may be produced by the same immunologic mechanism but be of unequal importance.

In the hereditary group of allergic diseases the cutaneous reactions obtained by testing with allergens are almost always mediated by the skin-sensitizing antibody which is present in both blood and tissues. An outstanding feature of the skin-sensitizing antibody is its affinity for the skin and the mucous membranes. Passive sensitization of respiratory, gastrointestinal, and ophthalmic mucous membranes may be as easily accomplished as the passive sensitization of skin. There is, therefore, sound reason for cutaneous testing in allergic dis-

eases of the hereditary type, even though the symptoms are derived from the mucous membranes and not the skin.

Cutaneous tests yield the most satisfactory results in allergic disorders of the respiratory tract in which inhalant allergens (pollens, dusts, danders, molds) play the most important role. In patients with allergic intrinsic dermatitis, migraine, and gastrointestinal allergic disease—conditions in which foods are likely to play some etiologic role—the skin tests, although less reliable, have contributed enough information with sufficient frequency to make them a necessary diagnostic procedure. In patients with drug sensitivities, chronic urticaria, and vernal conjunctivitis, skin testing is disappointing.

The diagnosis of allergic diseases is not a function of a laboratory technician who possesses and uses a set of diagnostic allergens. Unfortunately, for the sake of good medical practice and the welfare of the unsuspecting patient, clinical laboratories offering skin testing services to determine allergic sensitivity are still in existence. The services of most of these laboratories are rendered without the supervision of a certified specialist in allergy or a licensed physician who has had adequate special training in allergy. Skin tests, even when performed under the best of conditions, entail risks of severe or fatal reactions. These risks are curtailed to a point of insignificance when the tests are performed under the supervision of a physician who is familiar with the patient's history and physical status. More important still is the need of training and knowledge to wisely interpret the results of the tests.

The establishment of certification of specialists in allergy under the American Board of Internal Medicine indicates the belief of representative national organizations that the diagnosis and treatment of allergic disorders require considerable training and skill. Even though a physician experienced in allergy may not have fully met the requirements of certification, he is able to offer to his patient a standard of service that cannot be approached by any laboratory. A diagnosis of an allergic disease based on the shallow security of a slight, questionable positive reaction to skin test, rather than the thorough appraisal of the patient as an entity, leads to inaccuracy in diagnosis and to ineffectual treatment.

LETTERS to the Editor . . .

THE DEVELOPMENTS of medicine in California during the last quarter century reflect in no small part the growth and vigor of the State Medical Association—a growth of sweeping dimension which called for sound legal guidance and advice during many decades. The fact that such guidance and advice was ably given is evident to all who pause to look. Our thanks go forth to Hartley Peart.

To catalog the work of Mr. Peart on behalf of medicine would take more time and space than is at my disposal. Only those who have prepared for and sat through long Council meetings lasting often from Friday evening to late Sunday night, long Executive Committee meetings called at almost any day and hour, House of Delegates sessions that spanned the clock, court-room sessions on medical problems that saw whole weeks go by, and endless legislative hearings in the State Capitol—only they have any concept of the arduous tasks he and his associates have performed.

The record of our state in medical leadership is his testimonial. May I voice the feelings of myself and many colleagues in placing on record this small tribute to his memory and the part he played so long and ably in the molding of our professional destiny. We are indeed fortunate that his able and modest associates are still at our legal helm. To Mr. Hassard, Mr. Smith and their staff our sympathy and appreciation.

Yours very truly,

L. H. GARLAND, M.D.

California MEDICAL ASSOCIATION

NOTICES & REPORTS

Executive Committee Minutes

Tentative Draft: Minutes of the 245th Executive Committee Meeting, San Francisco, November 17, 1954.

The meeting was called to order by Chairman Heron in Room 214 of the St. Francis Hotel, San Francisco, at 4:30 p.m., Wednesday, November 17, 1954.

Roll Call:

Present were President Morrison, Council Chairman Lum, Speaker Charnock, Auditing Committee Chairman Heron, and, ex-officio, Secretary Daniels and Editor Wilbur. Absent for cause, President-elect Shipman.

A quorum present and acting.

Present by invitation during all or a part of the meeting were Messrs. Hunton, Thomas, Clancy and Gillette of C.M.A. staff; legal counsel Howard Hassard; Health Insurance Consultant Rollen Waterson; Drs. Dwight H. Murray, James C. Doyle and Dan O. Kilroy, members of the Committee on Public Policy and Legislation; Messrs. Ben H. Read and Eugene Salisbury of the Public Health League of California; Dr. Murray Hunter Brown of Los Angeles County; Dr. Richard A. Young, medical consultant to the California State Department of Vocational Rehabilitation; William S. Smith, D.D.S., Robert O. Schraft, D.D.S., E. L. Hicok, D.D.S., and Messrs. Anthony J. Kennedy and John Rooks, representing the California State Dental Association.

1. *A.M.A. Committee on Rural Health:*

On motion duly made and seconded, it was voted to approve the recommendation of President Morrison that Dr. Henry A. Randel of Fresno be nominated for membership on the Council on Rural Health of the American Medical Association, to succeed the late Dr. J. Frank Doughty.

2. *Rollen Waterson Associates:*

(a) Mr. Waterson stressed the need to develop adequate health insurance coverage for individual subscribers, to meet competition and to lead the way in this field.

On motion duly made and seconded, it was voted to urge strongly to the Board of Trustees that it use all possible efforts to develop and improve its individual membership coverage so as to make it more highly acceptable to the public.

(b) Mr. Waterson reported the findings of a spot survey to determine the number of members of closed-panel groups who sought medical care from their personal physicians at their own expense. He also reported that a pilot study of public opinion of the medical profession was under way.

(c) A budget covering the final calendar quarter of 1954, totaling $15,323, was approved, with the provision that $2,000 additional be allowed for producing and distributing tape recordings for playing before hospital staffs throughout the state.

3. *University Programs:*

Dr. Murray Hunter Brown of Los Angeles County presented information on programs being undertaken by a state university through some of its departments and it was agreed that members of the

Executive Committee should use their efforts to determine what other organizations or groups might be similarly concerned with the direction of such activities.

4. *California Medicine:*

Editor Wilbur requested an opinion as to whether or not CALIFORNIA MEDICINE should devote all or a large part of one issue to a discussion of medical topics concerned with civil defense. On motion duly made and seconded, it was voted to recommend to the Editor that one issue of the journal should be devoted to such subjects, at the discretion of the Editor.

5. *California State Board of Nurse Examiners:*

On motion duly made and seconded, it was voted to request the Council Chairman to forward to the California State Board of Nurse Examiners the names of four nominees for the appointment of two such nominees to the Advisory Council to this Board.

6. *State Board of Medical Examiners:*

Suggestions for nominees for appointment to the State Board of Medical Examiners were considered and it was agreed to refer this matter to the Council.

7. *Legal Department:*

Mr. Hassard discussed a report submitted by a county medical society on the operations of an organization which is apparently operating outside the scope of present laws in securing corneas for transplants. On motion duly made and seconded, it was voted to ask Mr. Hassard to confer with the Attorney General in this matter.

8. *Public Policy and Legislation:*

Drs. Schraft, Hicok and Smith, and Messrs. Kennedy and Rooks, representing the California State Dental Association, discussed proposed legislation, which would create one department of the state government to take over the functions of the present Department of Public Health, the Board of Medical Examiners, the Board of Dental Examiners and possibly other boards dealing with the healing arts.

Mr. Hassard discussed this proposal and it was duly moved, seconded and voted to approve this type of legislative proposal.

9. *State Bureau of Vocational Rehabilitation:*

Dr. Richard A. Young, medical consultant to the State Bureau of Vocational Rehabilitation, discussed the import of the new amendments to the Federal Social Security Law, dealing with total and permanent disability. He expressed his desire to work with the medical profession in the administration of this law. Dr. Young also urged the Committee to promote in every way possible the participation of physicians in community affairs, especially where questions of health and medical care are involved.

10. *Public Relations:*

(a) A communication from a member relative to establishing a series of radio programs was discussed and ordered transmitted to the Public Relations Department for conferences with the radio station administration.

(b) Mr. Clancy reported that more than 1,000,000 pamphlets in the current series provided by the Public Relations Department have already been distributed.

(c) Dr. Daniels reported on the Conference on Physicians and Schools held in Fresno November 12 and 13 and commented favorably on the interest and cooperation manifest by school and public health representatives present. A second such conference is planned in about two years.

11. *Executive Committee Meetings:*

On motion duly made and seconded, it was voted to hold dinner meetings of the Executive Committee on the second Wednesday of each month in San Francisco, with the exception of those months when the Council would meet at approximately the same time.

Adjournment:

There being no further business to come before it, the meeting was adjourned at 11:45 p.m.

IVAN C. HERON, M.D., *Chairman*
ALBERT C. DANIELS, M.D., *Secretary*

In Memoriam

HAMMACK, ROY W. Died in Los Angeles, September 20, 1954, aged 68, of leukemia and phlebitis. Graduate of Johns Hopkins University School of Medicine, Baltimore, Maryland, 1911. Licensed in California in 1916. Doctor Hammack was a member of the Los Angeles County Medical Association.

✤

HUGHES, JONES R. Died in Los Angeles, September 7, 1954, aged 51, of coronary thrombosis. Graduate of the University of Pennsylvania School of Medicine, Philadelphia, 1933. Licensed in California in 1934. Doctor Hughes was a member of the Los Angeles County Medical Association.

✤

MOLONY, CLEMENT J. Died in Los Angeles, December 4, 1954, aged 45. Graduate of Washington University School of Medicine, St. Louis, Missouri, 1934. Licensed in California in 1934. Doctor Molony was a member of the Los Angeles County Medical Association.

POPE, WILLIAM H. Died in Sacramento, December 6, 1954, aged 75. Graduate of the Medico-Chirurgical College of Philadelphia, Pennsylvania, 1907. Licensed in California in 1920. Doctor Pope was a member of the Sacramento Society for Medical Improvement, a life member of the California Medical Association, and an associate member of the American Medical Association.

✤

SOMMERS, IGNATIUS G. Died in Los Angeles, August 18, 1954, aged 60, of lymphosarcoma. Graduate of Medizinische Fakultät der Universität, Wien, Austria, 1919. Licensed in California in 1945. Doctor Sommers was a member of the Los Angeles County Medical Association.

✤

WILSON, CARL G. Died in Palo Alto, December 1, 1954, aged 74. Graduate of the College of Physicians and Surgeons, San Francisco, 1902. Licensed in California in 1902. Doctor Wilson was a member of the Santa Clara County Medical Society.

APPLICATION FOR HOUSING ACCOMMODATIONS

FOR YOUR CONVENIENCE in making hotel reservations for the coming meeting of the **California Medical Association,** May 1-4, 1955, in San Francisco, hotels and their rates are at the right. Use the form at the bottom of this page, indicating your first and second choice. Because of the limited number of single rooms available, you will stand a much better chance of securing accommodations of your choice if your request calls for rooms to be occupied by two or more persons. **All requests for reservations must give definite date and hour of arrival as well as definite date and approximate hour of departure; also names and addresses of all occupants of hotel rooms must be included.**

ALL RESERVATIONS MUST BE RECEIVED BEFORE: APRIL 15, 1955

NOTE: ***The House of Delegates will convene at the Sheraton-Palace Hotel; all scientific sessions and exhibits will be at the Civic Auditorium.***

Eighty-fourth Annual Session
CALIFORNIA MEDICAL ASSOCIATION

San Francisco, California
MAY 1-4, 1955

HOTEL ROOM RATES*

	Single	Double	Twin Beds	Suites
SHERATON-PALACE HOTEL Market and New Montgomery	7.50-15.00	10.00-17.50	10.50-18.00	19.00-65.00
ST. FRANCIS HOTEL Powell and Geary	9.00-20.00	11.00-16.00	13.00-22.00	24.00-40.00
SIR FRANCIS DRAKE HOTEL Powell and Sutter	8.50-14.00	10.50-16.50	13.00-19.50	25.00-36.00
CLIFT HOTEL Geary and Taylor	10.00-15.00	10.00-18.00	13.00-18.00	18.00-35.00
PLAZA HOTEL† Post and Stockton †Deposit required	6.00-7.00	.....	9.00-10.00	.....
STEWART HOTEL Geary and Powell	4.50-8.00	6.50-10.00	7.00-12.00	12.00-17.00
WHITCOMB HOTEL Market and Eighth	5.00-9.00	7.00-11.00	8.00-12.00	18.00-25.00
ALEXANDER HAMILTON O'Farrell and Hyde	6.00-11.00	8.00-13.50	8.50-14.00	12.00-30.00

***The above quoted rates are existing rates but are subject to any change which may be made in the future**

CALIFORNIA MEDICAL ASSOCIATION
450 Sutter Street—Room 2000
San Francisco 8, California

Please reserve the following accommodations for the 84th Annual Session of the California Medical Association, in San Francisco, May 1-4, 1955.

Single Room..... Double Bedded Room..... Twin Bedded Room.....

Small Suite..... Large Suite..... Other Type of Room.....

First Choice Hotel..... Second Choice Hotel.....

ARRIVING AT HOTEL (date).....Hour:.....A.M.....P.M. { Hotel reservations will be held until

Leaving (date).....Hour:.....A.M.....P.M. { 6:00 P.M., unless otherwise notified

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Therefore, please include the names of both persons for each double room or twin bedded room requested. Names and addresses of all persons for whom you are requesting reservations and who will occupy the rooms asked for:

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(Individual Requesting Reservations—Please print or type)

Name..... County.....

Address..... City and State.....

WOMAN'S AUXILIARY
TO THE CALIFORNIA MEDICAL ASSOCIATION

NATIONAL CONFERENCE HELD IN CHICAGO

THE ELEVENTH ANNUAL CONFERENCE of state presidents, presidents-elect and national committee chairmen of the Woman's Auxiliaries, held in Chicago in November, had as its theme "Leadership in Community Health." Representing California were Mrs. Carl Burkland, National Secretary; Mrs. Matthew Hosmer, state president-elect; and your correspondent.

Panel discussions by the Auxiliary members, as well as talks by guest speakers from the A.M.A., all stressed the importance of taking an active interest in health education and service on the community level. We were reminded that "As a physician's wife is his personal helpmeet, she is likewise his community envoy and representative; to her rightfully belongs leadership in health education and community health service."

* * *

OUR PROGRAMS STRESS SELF-EDUCATION

In order to take an active and intelligent interest in the health needs of their communities, our members must first be well-informed themselves. Our state program chairman, Mrs. Lawrence Custer of San Francisco, has planned and coordinated a well-balanced program for the counties, with emphasis on such topics as mental health, civilian defense, nurse recruitment, health legislation and voluntary health insurance plans. Several of our counties are having Mr. Ben Read, executive secretary to the California Public Health League, as their guest speaker during the year. He gives our members good advice on voting intelligently and stresses continued vigilance against the passage of bills that might be detrimental to the medical profession.

Cooperating in planning programs on legislation for the year is Mrs. Joseph Stout, Jr., of Pasadena, state legislative chairman. Much effective study is done by our Auxiliary study groups, where panels and round-table discussions as well as talks by guest speakers keep our members informed about legislative trends on state and national levels.

INTERESTING NEW PROJECT IN LOS ANGELES COUNTY

In every county, the Auxiliary tries to plan at least one joint dinner meeting with the Medical Society; usually they meet together on the night that the president of the California Medical Association makes his official visit.

The Los Angeles County Auxiliary has gone one step farther this year. Its president, Mrs. Elaine G. Cooper, has created the Caducean Section, which sponsors a series of dinners followed by lecture and film programs. Physicians show their own travel pictures and make their own commentaries, thus adding a personal touch to the programs. Aside from the cultural and educational benefits, physicians and their families are finding the meetings to be an excellent means of becoming better acquainted. We heartily recommend a similar plan to other counties.

* * *

DOCTORS TAKE TIME OUT FOR FUN, TOO

Most of our programs are serious, but once in a while we plan something purely for the entertainment of our members and their husbands. Sometimes these creative efforts gain nationwide fame. Such was the case in San Diego, after Mrs. James Phalen wrote the clever musical skit "There Is a Doctor in the House." This play has been presented to state and national conventions, and copies have been sent as far as Iowa, Indiana and Alabama. The group makes no charge for the script, but does specify that the skit must be presented for a nonprofit event.

Also popular throughout the state are costume dances for doctors and their wives; Kern County has a masquerade party every spring, and the Sonoma County Sono-Medics Sirkus has become a pleasant tradition. Fresno, too, has a gay spring dinner-dance complete with songs, dances and skits. Riverside County annually has a special party for the doctors; this year, the five-scene skit "Medical School Daze" was the featured entertainment.

Many of the annual parties bring tidy profits to the Nursing Scholarship Fund. In Placer-Sierra-Nevada, for instance, the October dinner-dance was as profitable as it was festive. Humboldt County, too, has an annual Christmas Ball which nets as high as $1,700 for the Nurses' Fund there. We'll tell you about other money-making parties next time.

MRS. FREDERICK J. MILLER, *President*

NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

The Seventh Annual **Mid-Winter Radiological Conference,** sponsored by the Los Angeles Radiological Society, will be held at the Ambassador Hotel in Los Angeles, on Saturday and Sunday, February 26 and 27, 1955. Conference reservations may be made through the chairman, Richard A. Kredel, M.D., 65 North Madison Avenue, Pasadena.

NAPA

At the annual meeting of **Napa County Medical Society** the following officers were elected: Dr. Wrenshall Oliver of Imola, president, succeeding Dr. Harold E. James of Sanatarium; Dr. Donald Marchus, Napa, vice-president; and Dr. Robert Ashley, Napa, secretary-treasurer, succeeding Dr. Merle F. Godfrey, Napa. Dr. Walter Brignoli of St. Helena was reelected a delegate to the California Medical Association, and Dr. Donald Marchus was reelected an alternate.

SAN FRANCISCO

The San Francisco Chapter of the **Pan-American Medical Association** held its annual dinner meeting honoring the local consular representatives and visiting Latin American and Canadian physicians and students at the Bohemian Club on November 27. Dr. Marius A. Francoz presided. Newly elected officers for 1955 are: Dr. Ralph A. Reynolds, president; Dr. Berthel H. Henning, first vice-president; Dr. Leonard Barnard, second vice-president; Dr. Knox H. Finley, treasurer; and Dr. Fernando G. Gomez, secretary.

SOLANO

Dr. Melvin A. Schmutz recently was elected president of the **Solano County Medical Society,** succeeding Dr. Herbert L. Joseph; Dr. Sam Nesting was elected vice-president and Dr. John C. Miller was elected to succeed Dr. Robert L. Garrett as secretary-treasurer.

SONOMA

Dr. Andrew E. Thuesen, Santa Rosa, was named president-elect of the **Sonoma County Medical Society** at its annual meeting, December 7. Dr. Robert S. Westphal succeeded Dr. William J. Rudee as president of the Society. Dr. Frank E. Lones was reelected secretary-treasurer.

GENERAL

At the annual meeting of the **California Society of Internal Medicine,** held at the Ahwahnee Hotel in Yosemite National Park on October 2, 1954, the following officers were elected for 1954-55: Dr. Paul I. Hoagland, Pasadena, president; Dr. George K. Wever, Stockton, vice-president; and Dr. James H. Thompson, San Francisco, secretary-treasurer.

The Councilors elected were: Dr. Walter P. Martin, Long Beach; Dr. William C. Mumler, Los Angeles; Dr. Charles A. Noble, Jr., San Francisco; Dr. Roy A. Ouer, San Diego; Dr. Robert L. Smith, Jr., San Francisco; and Dr. Arthur R. Twiss, Oakland.

* * *

Harold D. Chope, director of public health and welfare, San Mateo County, was elected president of the **California Conference of Local Health Officers** at the conference's eighth annual meeting held in Bakersfield November 15 and 16. Dr. Chope succeeds Dr. James C. Malcolm, health officer of Alameda County.

Dr. Edward Lee Russell, Orange County health officer, was elected vice-president and Dr. Ellis D. Sox, San Francisco health officer, secretary.

* * *

The California Society of Allergy wishes to extend an invitation to any member of the California Medical Association who has an interest in allergic diseases. The Society is dedicated to the advancement in scientific knowledge of allergic diseases and to the support of those institutions and foundations devoted to the dissemination of such knowledge and research.

Information concerning membership in the Society may be obtained by addressing Ben C. Eisenberg, M.D., Secretary-Treasurer, California Society of Allergy, 2680 Saturn Avenue, Huntington Park.

* * *

The Council on Undergraduate Medical Education of the **American College of Chest Physicians** has offered three cash awards to be given for the best contributions, prepared by any undergraduate medical student studying for a degree in medicine, on any phase of the diagnosis and treatment of chest diseases (heart and/or lungs).

The first prize will be a cash award of $250. Second prize will be $100 and third prize $50. The three winners will also receive a certificate of merit. The winning contributions will be announced at the 21st Annual Meeting of the American College of Chest Physicians, to be held in Atlantic City, New Jersey, June 2-5, 1955. Deadline for submitting manuscripts is April 10, 1955. Further information may be obtained from Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

* * *

The American Orthopsychiatric Association will hold its 32nd Annual Meeting at the Hotel Sherman, Chicago, on February 28, March 1 and 2, 1955. Approximately 100 scientific papers will be presented by psychiatrists, psychologists, social workers, educators, sociologists and anthropologists. There will be all-day sections on Childhood Schizophrenia, Child Development and Psychotherapy with Children.

POSTGRADUATE EDUCATION NOTICES

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

In San Francisco:

Electrocardiography for Beginners—January 31 to February 4.

Advanced Electrocardiography—January 31 to February 4.

Therapy of Cardiovascular Diseases—January 31 to February 4.

Atomic Energy Medicine—February 24 to February 27.

Course for General Practitioners—March 7 to March 11.

Symposia on Psychosomatic Medicine—March 23, 30, and April 6.

Recent Advances in Internal Medicine—April 18 to April 22.

Pediatric Conference—September (dates to be announced later).

Conference on Applied Therapeutics—October 17 to October 19.

Conference on Gynecology and Obstetrics—October 20 and October 21.

Ophthalmological Conference—December 5 to December 9.

In East Oakland:

Medicine for General Practitioners—Tuesday evenings, September 20 to December 6.

In Berkeley:

Postgraduate Conference—Wednesday evenings, September to December (dates to be announced later).

In San Mateo:

Evening Lectures in Medicine—Thursday evenings, September 22 to December 15.

Contact: Office of Medical Extension, University of California Medical Center, San Francisco 22.

STANFORD UNIVERSITY SCHOOL OF MEDICINE, SAN FRANCISCO

Spring Conference in Ophthalmology—March 21 through March 25.

Contact: Office of the Dean, Stanford University School of Medicine, 2398 Sacramento Street, San Francisco 15.

UNIVERSITY OF SOUTHERN CALIFORNIA AT LOS ANGELES

The Medical Extension Education Division of the University of Southern California School of Medicine announces:

Four courses to be offered for twelve weeks starting January 6, 1955, as follows:

*No. 841 Course—Basic Principles of Cardiorespiratory Physiology and Their Clinical Application.

No. 873 Course—Cardiac Resuscitation, sponsored by the Los Angeles County Heart Association.

No. 864 Course—Pediatric Clinics for General Practitioners.

No. 846 Course—Seminars in Advanced Gastroenterology.

For application blanks or other information call or write to Division of Medical Extension Education, University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 33 (Capitol 5-1511).

*Course No. 841 is restricted to specialists; all other courses are open to graduates of Grade A medical schools who have completed an approved internship.

CHILDREN'S HOSPITAL SEMINARS

The Management of Metabolic Disturbances Commonly Encountered in Practice—January 22, 1955.

The Allergic Dilemma—February 26, 1955.

Infections and Their Management—March 26, 1955

Accreditation by the Board of General Practice has been granted. Gertrude F. Jones, M.D., Chairman, Medical Alumni Committee, Children's Hospital, 3700 California Street, San Francisco 18.

SAN FRANCISCO HEART ASSOCIATION

Twenty-sixth Annual Postgraduate Symposium on Heart Disease at the St. Francis Hotel, San Francisco, October 5, 6, 7, 1955.

For information contact Gladys Taylor Daniloff, Executive Director, San Francisco Heart Association, 604 Mission Street, San Francisco 5, Calif.

CALIFORNIA MEDICAL ASSOCIATION, POSTGRADUATE ACTIVITIES INSTITUTES

Southern Counties—Tennis Club, Palm Springs—January 27-28, 1955.

North Coast Counties—Santa Rosa—February 3-4, 1955.

West Coast Counties—Santa Barbara—February 17-18, 1955.

San Joaquin Valley Counties—Yosemite—April 13, 14, 15, 1955.

Sacramento Valley Counties—Cal-Neva—June 16-17, 1955.

Contact: C. A. Broaddus, M.D., Director of Postgraduate Activities, P.O. Box AI, Carmel, California.

Medical Dates Bulletin

This bulletin of the dates of postgraduate education assemblies and the meetings of various medical organizations in California is supplied by the Committee on Postgraduate Activities of the California Medical Association. In order that they may be listed here, please send communications relating to your future medical or surgical programs to: C. A. Broaddus, M.D., P.O. Box AI, Carmel, California.

JANUARY MEETINGS

January 17-28—Research Study Club of Los Angeles: Twenty-fourth Annual Midwinter Clinical Convention in Ophthalmology and Otolaryngology. Mr. H. M. Nickerson, manager of the Elks Club, 607 South Parkview Street, Los Angeles 57.

January 21-22—American College of Surgeons: Palm Springs.

January 26-28—American Board of Orthopedic Surgery: Part II, Oral and Written, Examination, Los Angeles. Final date for filing application for Part II was August 15, 1954. Dr. Harold A. Scofield, 122 S. Michigan Avenue, Chicago, Ill.

* * *

California Medical Association: Annual Session, May 1-4, 1955, San Francisco.

American Medical Association: Annual Session, 1955, Atlantic City, June 6-10; Clinical Session, 1955, Boston, November 29-December 2.

California Society of Internal Medicine: October 1, 1955, Biltmore Hotel, Santa Barbara, Mildred D. Coleman, Assistant Secretary, 384 Post Street, San Francisco 8.

INFORMATION

Malpractice Claims Prevention Programs

Are They Worth While?

HOWARD HASSARD, *Esq.*
Of Peart, Baraty & Hassard, San Francisco

ARE MALPRACTICE CLAIMS prevention programs worth while? Suppose we break that question into several pieces and examine each in an effort to discern the underlying factors that cause the question to be posed.

First: "What is to be *prevented*?"

To answer this, a brief historical review is appropriate. Regan tells us that in the decades between 1900 and 1940 there was a 540 per cent increase in the number of malpractice cases that reached the appellate courts in the United States as a whole. In the year 1940 the total number of such cases was 33. In 1953 there were 32.

These years were picked at random. They indicate a plateau, which practical experience confirms. The volume remains at least five times the 1900 incidence.

Dr. Regan's statistics demonstrate the tremendous rise in the incidence of malpractice suits in the past fifty years. Further, a little research in a law library discloses that the increase is perhaps more noticeable in the largest metropolitan centers, but that it is by no means confined to any one area. It cannot be localized.

Lest physicians assume that they are being singled out by the public for special torture, it must also be understood that all forms of personal injury litigation have dramatically increased since World War I. Mass production of the automobile has wrought many changes, one of them being increased frequency of accidental injuries or death and increased resort to law for redress.

When people become suit-conscious in general, they tend to think in terms of legal action for any and all real or fancied grievances. Fifty years ago a guest in a home would consider it ungentlemanly to sue his host because after the third martini he wandered through a plate glass window. Nowadays suits of this type are not too uncommon.

With the public litigation-conscious, there is a tendency to commence legal action, not only when warranted, but also when there is just a bare chance of recovery; sometimes even when there is no legitimate cause for complaint.

Within the field of professional liability, the main activity that can be "prevented" is the fraudulent or false or vindictive or long-shot suit that is not based on just cause. The meritorious action not only cannot be prevented, but ought not be impeded. However, to separate the sheep from the goats and thereby reduce the incidence of nuisance claims would drastically curtail professional liability actions, and of itself is a justifiable reason for a claims prevention program.

Second: "What is malpractice?"

"Malpractice" is the commonly used term to describe the liability at law of physicians and surgeons for torts committed during the course of their practice. Properly stated, it is "professional tort liability." A "tort" is a violation of one's duty to use reasonable precaution for the safety of others, resulting in an injury to another.

By law, we are all obliged at all times to be reasonably careful of the safety of others. If one of a group of people seated together in a room, for example, should suddenly jump up, knocking over his chair in the process, and if the chair injured the person sitting behind him, he might find himself the defendant in a tort action for having failed to use ordinary care.

As applied to physicians, the law requires that each physician possess the average skill found amongst fellow-practitioners doing the same work in his own community, and that he at all times exercise ordinary prudence and thoughtfulness in the application of his skill to his patients. The failure to live up to these obligations is called "malpractice."

The ordinary personal injury suit against the average person involves his pocketbook only. Hence, if he is adequately insured he gives the fact of a suit against him very superficial concern.

But to a physician, or any other professional man, a professional liability suit involves something else that is much deeper, much more important. His professional reputation, his very livelihood, his pride and his self-respect are all at stake. In his mind, it is an accusation akin to a charge of dishonorable conduct. It is humiliating.

Therefore, we must not look solely to the financial aspects of malpractice.

Each physician, in order to avoid the humiliation of a liability suit, must become thoroughly familiar with the various rules of law, that together, constitute the law of malpractice. He must intimately know the rules of the game.

Presented before the 1954 Annual Meeting of the Medical Society Executives' Conference, San Francisco, June 21.

Medical schools are not law schools. Hence the practicing physician must acquire his knowledge of the law that governs him after he is in practice, and he may acquire this knowledge either haphazardly or systematically. He will pick up his concepts either on a hit-or-miss basis from dubious sources or he will acquire it in an orderly fashion from teachers who know at least as much as the student.

A systematic, well-organized professional educational program in the field of malpractice has the possibility of achieving a tremendous reduction in the incidence of malpractice claims and suits. By educating physicians to their legal responsibilities and to the required conduct in carrying out those responsibilities, approval of the law, of the public and of patients may be obtained and maintained.

Malpractice has another most important facet that must be understood in any discussion of a claims prevention program.

All physicians today are, or ought to be, insured against professional liability. This insurance, however, is far from the ordinary run-of-the-mill public liability coverage.

It is true that the legal theory underlying responsibility for running down a pedestrian or for burning a patient with an ultraviolet lamp are one and the same; but beyond that, all resemblance ceases. From the moment of knowledge, the investigation, claims analysis, preparation for defense, and defense of an automobile personal injury case are standardized, not too difficult to master, and fit into the ordinary operations of any insurance claims department or law office.

The investigation, the claims analysis, the preparation for defense, and the defense against a malpractice claim are not in any way comparable to these factors with regard to other personal injuries. An investigator must know enough about the practice of medicine to be able to know what to investigate when a claim of malpractice has been made. The analysis of the results of the investigation requires expert medical judgment. The defense of a malpractice case in court involves specialized training in this field. The rules of evidence and the substantive rules of law are different than in the ordinary personal injury case. The lawyer must understand the medical aspects thoroughly, so that he can communicate in ordinary English to the judge and jurors the issues and facts involved.

Recently, I appeared in Federal Court at Salt Lake City, and while awaiting the commencement of our trial, I sat in the courtroom and observed the case that preceded us, which was a suit by the Navajo Nation against the United States for damages resulting from the destruction of Navajo horses by agents of the United States Indian Service. The witnesses were all Navajo Indians who could not speak English and interpreting was necessary. Each question was translated by the interpreter into Navajo; when the witness replied, the interpreter translated the answer. The net result was that the trial took twice as long as it would have if court, jury, counsel, and witnesses all spoke in a commonly understood tongue.

This is an extreme example, but a malpractice trial is similar. Medical terms must first be understood by counsel, and then converted into language understood by judge and jurors.

It is obvious that insurance companies that have a few malpractice policies outstanding in a community cannot afford to set up separate specialized malpractice claims departments or employ attorneys who specialize in malpractice defense. Premium volume is too small to warrant tailor-made or custom handling. To justify expenditure of funds for special treatment of malpractice policies, there must be a substantial volume, which means all or most of the physicians over a large area.

An insurance carrier, unless it has a large volume, cannot afford the further expense of a specialized prophylaxis or prevention program. Fire insurance companies, with all of their business at risk, can afford to spend substantial sums of money in fire prevention programs. Workmen's compensation insurers, with hundreds of thousands of employees insured, can afford to spend money on safety programs. But an insurer with a few hundred scattered physicians insured simply cannot do so.

One essential of insurance is spread of risk. The whole field of physicians' professional liability in the United States is limited to approximately 160,000 physicians. If one company insured all, the insurance base would be minor, as compared to twenty million automobile owners, or fifty or sixty million homes, or the sixty to seventy million people covered by workmen's compensation.

Hence, one of the inherent problems in malpractice insurance is the limited market and the consequent limited ability of any one carrier to conduct the equivalent of a safety program.

Third: "What is a *program*?"

In northern California, 23 county medical societies now have professional liability insurance contracts with the same insurance carrier, American Mutual Liability Insurance Company. In all, close to four thousand physicians are participating. While each county has it own group contract, the program is substantially the same from Fresno in the south to Siskiyou in the north.

Each county has a medical committee. In the early stages of each claim against a physician, the facts are fully investigated by claims representatives of the insurer, who devote their entire time to this type of work. As claims adjusters, they are "specialists" in professional liability work.

When the case is investigated, the facts are then submitted to the society's medical committee. The members of the committee discuss and debate the case, sometimes call for more investigation, sometimes ponder their decision at length, on other occasions reach a conclusion fairly rapidly.

In any event, the committee satisfies itself that it has considered all the material facts, and then recommends either—

1. That the claim has merit and that the claimant should be fairly compensated; or

2. That the facts do not disclose any medical dereliction on the part of the accused physician, and that the case should be defended.

To date, in each instance the insurance carrier has abided by the recommendations of the appropriate committee.

The functioning of the society's committee does not, however, terminate with recommended action. If it has recommended that the case be defended, the members of the committee then actively and voluntarily assist in the preparation of the defense and in the actual trial of the case. To the defense attorney, this is of invaluable aid. Incidentally, it reduces the cost of defense substantially.

Finally, the members of the various medical committees also appear before various professional audiences, and from their experiences undertake to explain to the practicing physician the legal pitfalls that beset a doctor and the conduct which should be adhered to to avoid legal liability.

The physicians who serve on these committees obtain "occupational experience." They know from having experienced specific cases what the problems are, and what information a physician needs in order to conduct "good practice" rather than "bad practice."

Admittedly, the program in California is far from perfect. A great deal more could be done, and should be done, to inform all physicians of their legal obligations, and to enable them to avoid the humiliation of a malpractice suit.

More manpower than has been available to date is no doubt needed. But at least we believe that this program is a sensible beginning. We feel that malpractice claims are intelligently analyzed, and that time and money is not wasted in endeavoring to defend the indefensible. On the other hand, unwarranted claims are discouraged in that nuisance settlements are not made. If the case is unjust it is defended; it is not settled, no matter how cheap it can be bought.

Physician participation in the trial of cases is obtained on a voluntary cooperative basis, and above all the physicians who serve on the medical committees become experienced in and aware of the problems involved, and are able to do missionary work amongst their colleagues.

It takes years for the results of such a program to become really measurable. We feel that at least ten years, and probably fifteen years, must elapse—and we are now only in the fifth full year—before any reliable inventory can be made.

However, the results to date indicate to us who are close to the picture that we have at least halted that steady increase in the incidence of malpractice claims and suit, noted by Dr. Regan, that commenced early in the century and that has continued without interruption for fifty years.

There are a few specific observations that are somewhat beside the principal theme of this address, but that are most important and ought not to be ignored in considering the value of any prevention program.

1. The incidence of malpractice claims is in inverse ratio to the degree of personal relationship between physician and patient. The more impersonal and aloof a physician is, the more critical the patients are bound to be.

2. The confidence of a patient in his physician is rudely jolted when another physician makes sarcastic or derogatory comments. The roots of many malpractice cases are embedded in such remarks as "What butcher performed that operation?" or, "How in the world could he have missed it?" The physician, like all of us, must sell himself. The art of salesmanship is not easily acquired and the amateur usually does the wrong thing. He builds himself up by knocking others. Actually, that is poor salesmanship. The expert salesman ignores his competitor and concentrates on establishing confidence in himself.

3. Even the poor have pride, and a certain way to wound deep personal pride and self-respect is for a physician to send a bill that his patient cannot pay and in so doing humble the patient to the point where he has to ask for charity. Many a malpractice case has its roots in the thoughtless handling by a physician of the financial side of his practice. The bill does not have to be exorbitant to cause anger and resentment; it can be reasonable, but if it humiliates, resentment is immediately aroused. A little tact and a little inquiry before billing could save many a headache.

4. Lawyers soon learn not to believe everything that their clients tell them. People have a habit of stating as fact that which they would like to believe, not the cold cruel reality. Many physicians find it difficult to realize that the tales their patients tell them may not necessarily be true. Consequently, a patient who is shopping will tell a physician a tall story about treatment that he received from another doctor; the physician accepts it as true, and comments accordingly; next year, he is in court.

5. Inherently, malpractice prevention is entwined with malpractice insurance. The insurance obtained

must be adequate and the carrier *interested*. Insurance is a commodity, it comes in different prices and packages and is produced to fit a market. If one buys the cheapest policy, one gets exactly what is deserved—the lowest quality. For physicians to buy malpractice insurance solely on the basis of price, is, to my mind, foolish. Recently, a California physician cancelled his group coverage because he could save ten dollars elsewhere. He said, "We feel that with today's competitive prices we have to be on the lookout for savings." So will his carrier when he is faced with a claim of malpractice. Then he will learn. A malpractice prevention program and basement bargain sales are incompatible with each other. A safety program costs money, whether it involves your home, factory or profession.

These, then, are some of the reasons why a prevention or safety program is worth while and why to undertake it requires group, rather than individual, action.

If by now anyone doubts the need for control of "malpractice" suits, allow me to quote from the May 1954 issue of the American College of Radiology News Letter:

"There are many reasons why the entire medical profession today finds itself in the same position that radiology was in 20 years ago. Here are some of the recent causes that have gone into the pot to make hospital and physicians' liability insurance even more undesirable from the underwriters' standpoint: increased demand for medical and hospital care; legislation increasing hospital liability; specialization, excessive fees; increased public 'claim consciousness'; bad hospital public relations; hospitalization insurance; dollar 'madness'; court interpretations broadening liability in this field; increased costs of legal work and investigation.

"The most potent factor, however, is the ever spiraling inflation and dollar devaluation—to which there seems to be no end—and which has resulted in fantastic judgments being rendered by juries in personal injury and malpractice suits."

Again, quoting from the same article in connection with the problem of lack of interest by insurance carriers:

"Some few of the companies are reluctantly writing business at the Bureau rates. One company will write for only their own agents and will cover x-ray therapy, providing the assured has been certified by the American Board of Radiology or is a member of the American Roentgen Ray Society or the Radiological Society of North America. Another company will write for its own agents only and will not write or renew existing policies for brokers. In addition, the applicant must promise the company all of his insurance business as collateral."

Physicians are in jeopardy until the insurance industry again is *interested* in insuring them. This will not occur unless and until the risk in professional liability insurance is lessened materially. The *risk* won't decrease of its own accord. A real, vigorous and widespread but grass roots program—by the medical profession itself—to educate its members to their legal duties, to advise and assist when trouble brews, and to fight relentlessly all unjust claims, is the only prudent course of action, if disaster is to be avoided.

111 Sutter Street, San Francisco 4.

THE PHYSICIAN'S *Bookshelf*

SMOKING AND CANCER—A Doctor's Report. Alton Ochsner, M.D. Julian Messner, Inc., New York, N. Y., 1954. 86 pages, $2.00.

There is an old saying that there is none so righteous as a reformed rake. Alas for that adage, the author of this work states, "I myself have never smoked." Perhaps not cigarettes, but his pages surely smoke like fire and brimstone.

After an introduction by Professor Evarts Graham—marred, alas, by several errors—the author plunges into a cascade of 16 indignant chapters studded with even larger errors.

"Cigarettes cause cancer." This bald and scientifically unproven statement—unproven insofar as human lung cancer is concerned—opens chapter one. "In 1954 some 23,000 Americans will die from lung cancer." Dr. Ochsner means respiratory tract cancer; the fact that only about 75 per cent of *respiratory tract* cancer is due to *bronchial* carcinoma is omitted.

Besides causing cancer of the lung, smoking is credited with leading to impotency in men and sterility in women, heart disease, cerebral hemorrhage, indigestion and asthma. No scientific data are offered to support these dogmatic assertions. Instead, the author relies on material accumulated by volunteer workers for a national welfare organization, data based on "reported" (not verified) cancer deaths in a sample of only 5 per cent of the population group under study. These data, if correct, do show an association between reported lung cancer deaths and reported smoking habits, but the figures differ widely from those recently published by Doll and Hill as a result of a careful prospective survey of cancer deaths in British physicians.

For example, the American Cancer Society figures "show" that "cigarette smoking is associated with an increase in the death rate from most of the common sites of cancer in men." However, the Doll and Hill figures show that cancer (outside the lung) is commoner in nonsmokers than in smokers.

Concerning tobacco carcinogenesis, Dr. Ochsner does state (p. 9) that "In our present ignorance we do not know exactly what happens during this process." He could have added "or whether the tobacco factor is more than a fractional one," for the simple reason that the very figures he leans upon so heavily show that at least 99,761 out of every 100,000 male heavy smokers (males from 50 to 70 who have smoked over one pack of cigarettes a day for 20 or more years) *do not die of lung cancer.* The Hammond-Horn data show, in their particular sample, an apparent risk of 27 deaths per 100,000 in nonsmokers; a risk four times as much in moderate smokers; and one nine times as much in heavy smokers. However, if limited to squamous cell cancers (the ones supposedly caused by cigarettes) the rate is still only about 200 per 100,000 heavy smokers ages 50 to 70.

The Graham-Wynder experimentally-produced mouse skin cancers are reported. To equal that exposure concentration in human lungs, your reviewer has been told that a distinguished Chicago pathologist calculated a man would have to smoke some 600,000 cigarettes a day for 64 years. Furthermore, in the Wynder mice, more females than males developed skin cancer; the ratio in human lung cancer is of course about 5 males to 1 female.

Space does not permit further comments on the data offered by Dr. Ochsner. However, a few quotes should permit the reader to gauge the tenor of the rest of this Catiline oration:

"As your cigarette burns, more than 200 chemical substances appear, flare up and vanish . . . it's somewhat like puffing on a small H-bomb."

"Cancer of the larynx has increased approximately as rapidly as lung cancer in the past twenty years." The scientific *facts* available to your reviewer show that this is not true.

"Tobacco . . . can cause . . . an ulcer (of the stomach). *Often* such ulcers become malignant." This indeed is news, or perhaps grade IV hysteria.

In calmer mood, the author mentions Doll's estimate that at least 20 per cent of lung cancer deaths in England in 1950 were attributable to causes other than smoking, that many investigators believe other air pollutants may be a greater factor, and that many heavy smokers do reach a ripe old age to die of nonnicotinogenous causes. He condemns the cigarette industry for its blatant advertising of two decades back and its evil domination of television today. This may well be true, but too bad to outdo Carrie Nation by his adding . . . "Would the measures I have suggested 'ruin' the tobacco industry? Hardly . . . not as long as there are smokers who will deliberately suffer *quadruple amputation,* rather than heed the warning to quit smoking." Gentle reader, how many *quadruple amputations* resulting from endarteritis obliterans due to nicotine addiction are there on scientific record? Please, Dr. Ochsner.

No one should consume excessive amounts of sugar, cream or butter, for obesity and heart disease are handmaidens. No one should abuse alcohol, caffeine or nicotine, for drug addiction and disease are cousins. Nor should suspicion and premature conclusions lead to intemperance in words and action. Have a smoke, Dr. Ochsner, just one—and relax.

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PROCEEDINGS OF THE FOURTH INTERNATIONAL CONGRESS OF THE INTERNATIONAL SOCIETY OF HEMATOLOGY—1952. Associate Editors: F. Jiminez de Asua, Buenos Aires; William Dameshek, Boston; and Sol Haberman, Dallas. Grune & Stratton, Inc., New York, 1954. 473 pages, $10.00.

This is a volume of collected papers presented before the International Society of Hematology in Argentina in 1952. The delay in publication is unfortunate but probably un-

avoidable. This is a similar but somewhat smaller book than that of the previous Proceedings. The papers are grouped in one of seven sections which included neuroendocrinologic regulation of hemotopoiesis and hemostasis, histochemistry and cellular ultrastructure, the leukemias, manifestations of radioactivity in hematopoietic organs and hemostasis, hemolytic disease, and hemorrhagic disturbances. The excellent papers are too numerous to mention individually. In addition to full length articles, many summaries of other papers are included. Many of the papers are in Spanish but English summaries are presented. While there are papers such as Farber's review of his group's experiences in the treatment of acute leukemia which would be of interest to the general physician and internist, the volume is primarily of interest to the clinical and laboratory hematologist.

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FUNDAMENTALS OF OTOLARYNGOLOGY—A Textbook of Ear, Nose and Throat Diseases—2nd ed. Lawrence R. Boies, M.D., Clinical Professor of Otolaryngology, Director of Otolaryngology, University of Minnesota Medical School. W. B. Saunders Company, Philadelphia, 1954. 487 pages, $7.00.

This second edition fulfills the author's statement that it represents a thorough revision of the first edition with some additions to bring the book completely up-to-date in this changing medical world.

The changes and additions comprise format, type, and an added chapter on bronchoscopy, bronchography, and pulmonary disease, information concerning the properties and uses of several of the better known antibiotics, the antihistamine drugs, and a paragraph about ACTH and cortisone. Of particular interest is a thought provoking addendum under the heading "The Possibilities of Transudate Disorders in Otolarnygology: Allergy, Autonomic Dysfunction and Endocrine Imbalance," by Jerome A. Hilger, M.D.

It is unfortunate that the revised edition retained several of the rather poor illustrations, some of which are of little, if any, real value.

The expanded indications for tracheotomy and the more frequent performance of this surgical procedure, make one wish that the author had at least mentioned the advantages of the horizontal or modified collar incision. The described vertical incision can offer no advantage except priority, and it frequently results in a disfiguring scar that taxes or defeats the most facile plastic surgeon.

The first edition was, and is, an excellent text for the undergraduate and the physician who is not a specialist in this field. This second edition is better than the first one and is recommended as one of those volumes which should be on hand and within easy reach.

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LEGAL MEDICINE. Edited by R. B. H. Gradwohl, M.D., Sc.D., F.A.P.H.A., Commander, M.C., USNR (Ret.), Director of the Police Laboratory, Metropolitan Police Department, St. Louis; First President, American Academy of Forensic Sciences. The C. V. Mosby Co., St. Louis, 1954. 1,093 pages, 222 illustrations, $20.00.

A fairly complete volume on the subject of legal medicine, with an impressive list of contributors who represent the leading authorities in the various phases of legal medicine.

The lists of references following each of the sections is a useful source for those interested in special inquiry into some single field of legal medicine.

While the various subdivisions of legal medicine are more thoroughly treated in individual volumes on the subject this book presents a good compilation of the more commonly needed information.

The purely medical aspects of the subject are as well or better treated elsewhere. The technical subjects of hair analysis identification, blood stains, blood grouping, alcohol determination, and alcohol intoxication are extremely well presented and of particular value. The section on Forensic Psychiatry makes informative and easy reading. The indexing is adequate, although at times disappointing.

On the whole the book is an authoritative general review of the subject, making a good single source for information.

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HYPERTENSION AND NEPHRITIS—5th ed, enlarged and thoroughly revised. Arthur M. Fishberg, M.D., Director of Medicine, Beth Israel Hospital, New York, Clinical Professor of Medicine, New York University Postgraduate Medical School. Lea & Febiger, Philadelphia, 1954. 986 pages, $12.50.

This well known book has undergone its first revision in 15 years. Several new chapters have been added including those on the treatment of essential hypertension and on diabetic glomerulosclerosis. Outstanding as before is its readability. The approach is generally a practical one for the clinician. The author in his preface states that the book is written for the practitioner and student, and it fills this role admirably. Both sides of controversial subjects are fairly presented. The specialist in cardiorenal disease will find the material valuable for review. An extensive bibliography is offered although recent references are not as numerous as might be expected. Despite the number of editions this book has undergone through the years, it is still outstanding in the field.

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LABORATORY AIDS IN ENDOCRINE DIAGNOSIS. Roberto F. Escamilla, M.D., Associate Clinical Professor of Medicine, University of California School of Medicine. Charles C. Thomas, Publisher, Springfield, Ill., 1954. 131 pages, $4.75.

There is a great need for a summarization of the many complicated laboratory procedures used in the diagnosis of endocrine and metabolic disease problems, and this book is written to fulfill this need. The basic design of the book is excellent. The various laboratory tests on blood, urine, hormone assays, special procedures such as radio iodine uptake, useful x-ray studies, vaginal smears, endometrial biopsy, semen examination and testicular biopsy are described in detail, including the preparation of reagents, the technique of the test, the normal range, and the diseases in which the test may be abnormal. Finally, in the last chapter, under the headings of the various endocrinopathies, such as pituitary disease, thyroid disease, adrenal disease, gonadal disease, etc., the most useful diagnostic tests as well as those occasionally helpful are listed, and the expected abnormalities are summarized. Thus the clinician may find at a glance the tests which may help him solve his diagnostic endocrine problems and a technique for doing that test.

Unfortunately, laboratory tests, and particularly the hormone assays, are not as precise as one would like them to be. The results may vary with the method used, the reagents used, the laboratory doing the test, the collection of the specimen, and other variables. Therefore, in order to use these laboratory procedures, one must know not only the name of the desired test, but also the variability of the procedure in the laboratory selected to run it. New methods for hormone assay are being developed which may make certain tests presently used obsolete. However, this book will provide for the internist, the general practitioner, and the laboratory director a clear outline of the indications and the techniques of the most useful procedures in the diagnosis of Endocrine Disease, which are at present generally available.